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# Archives of Neurology and Psychiatry

Vol. 2

SEPTEMBER, 1919

No. 3

## PERIPHERAL NERVE INJURIES CONCOMITANT TO GUNSHOT WOUNDS

PRELIMINARY REPORT ON THE DIAGNOSIS, OPERATIONS,  
PROGRESS AND RESULT OF TREATMENT IN  
FIVE HUNDRED AND TEN CASES

IRVING J. SPEAR, M.D.

Major, M. C., U. S. Army; Neurologist, U. S. Army General Hospital No. 6

AND

W. W. BABCOCK, M.D.

Lieutenant-Colonel, M. C., U. S. Army; Chief of Surgical Service,  
U. S. Army General Hospital No. 6

FORT MC PHERSON, GA.

Four hundred and nineteen patients with peripheral nerve injuries were admitted to U. S. General Hospital No. 6, between October, 1918, and May 1, 1919. Table 1 shows the actual number of each individual nerve that was injured, the operations which were performed on that nerve, and the number which up to May 1 had not been operated on. Table 2, independent of time, shows the number of the individual nerves that showed no improvement, the number that were improved or cured, as the result of operation and treatment or treatment alone, and the number that have not, since their admission to the hospital, returned for a second examination. Table 3 shows the actual number of nerves that were made worse, remained the same, were improved, much improved and the number that were cured; also the operations that brought about these results.

Most of the patients entered the hospital at least four months after the infliction of the wound, and a large majority were not operated on until at least six months after sustaining their injury. All patients, with one or two exceptions, were examined at least once before the operation. Operation was advised only in those cases in which the neurological examination justified the diagnosis of complete physiologic block. We considered physiologic block to be present when there existed total paralysis with atrophy of the affected muscles, complete reaction of degeneration, and in a mixed nerve loss of sensation.

TABLE 1.—NERVE OPERATIONS PERFORMED AND NUMBER NOT OPERATED ON IN U. S. ARMY GENERAL HOSPITAL NO. 6, FORT MCPHERSON, GA.

	Operations				Nonoperative or Not Examined Since Operation	Total
	Neurolysis	Hersage	Suture	Total		
7th.....	..	..	2	2	..	2
S. A. ....	1	..	..	1	..	1
C. P. ....	2	..	..	2	2	4
B. P. ....	3	6	2	11	15	26
C. ....	1	..	..	1	1	2
M. C. ....	3	4	3	10	6	16
M. S. ....	8	12	17	37	48	85
H. ....	8	15	43	66	37	103
M. ....	12	14	16	42	33	75
I. C. ....	1	..	4	5	6	11
R. ....	3	..	2	5	6	11
A. I. ....	..	1	1	2	1	3
P. I. ....	1	1	..	2	2	4
L. P. ....	..	..	..	..	1	1
S. ....	1	16	19	36	38	74
A. C. ....	..	..	1	1	7	8
E. P. ....	2	1	11	14	23	37
I. P. ....	..	2	2	4	7	11
I. S. ....	..	..	2	2	5	7
A. T. ....	..	3	8	11	3	14
P. T. ....	2	..	2	4	11	15
M. C. ....	..	..	1	1	..	1
Total.....	48	75	136	259	252	511

TABLE 2.—NUMBER OF PATIENTS THAT SHOWED IMPROVEMENT, NUMBER IMPROVED OR CURED AND NUMBER THAT DID NOT RETURN FOR EXAMINATION

	Operative					Nonoperative				
	Worse	Same	Improved	Much Improved	Cured	Same	Improved	Much Improved	Cured	Not Returning for Examination
7th.....	..	2	..	..	..	..	..	..	..	..
S. A. ....	..	1	..	..	..	..	..	..	..	..
C. P. ....	..	1	..	..	..	..	..	..	..	..
B. P. ....	..	..	8	3	..	..	6	5	1	3
C. ....	..	..	1	..	..	..	..	..	1	..
M. C. ....	..	4	1	4	1	1	..	..	1	..
M. S. ....	..	15	11	7	4	9	5	..	9	23
U. ....	1	33	24	5	3	6	6	..	4	19
M. ....	..	12	21	5	4	1	10	..	4	16
I. C. ....	..	4	1	..	..	2	1	..	..	3
R. ....	..	1	..	..	..	3	1	..	1	1
A. I. ....	..	..	..	1	1	..	1	..	..	..
P. I. ....	..	..	1	1	..	..	2	..	..	..
L. P. ....	..	..	..	..	..	1	..	..	..	..
S. ....	..	26	9	1	..	8	7	1	..	20
A. C. ....	..	1	..	..	..	1	..	..	..	6
E. P. ....	..	11	2	..	1	7	3	2	1	10
I. P. ....	..	2	1	1	..	..	2	1	..	4
I. S. ....	..	2	..	..	..	2	1	..	..	2
A. T. ....	1	7	2	1	..	..	1	..	1	1
P. T. ....	..	2	2	..	..	6	2	..	3	110
M. C. ....	..	1	..	..	..	..	..	..	..	..
Total.....	2	125	86	29	16	47	52	15	28	220
258					142					

TABLE 3.—OPERATIONS PERFORMED ON NERVES AND RESULTS

	Worse	Same	Improved	Much Improved	Cured	Total
Neurolysis.....	0	10	22	7	9	48
Hersage.....	1	16	39	12	7	75
Suture.....	0	100	31	4	1	136
Totals.....	1	126	92	23	17	259

We do not believe it is possible to foretell, even in the presence of symptoms of complete physiologic block, whether this was due to an interruption of continuity, or to the presence of scar tissue in a nerve. Operation was also advised in those cases that showed marked irritative phenomena, such as constant pain and very marked vasomotor and trophic disturbance in the absence of vascular involvement. In some cases there existed a doubt as to the necessity of operation, and these cases were reexamined in from two to six weeks before final advice was given.

A group of cases was examined in which loss of function was the prominent symptom; however, the motor disturbance was not due to neurological involvement, but was caused by injury to muscle, bone or joints. These cases were referred back to the surgical department with the diagnosis: "neurological examination negative."

There are two points of view from which the improvement and cure of a patient can be considered:

1. A patient may be considered improved, or cured, who has a beginning return of function, or almost complete return of function as the result of the education and increased power of adjuvant muscles, and of learning new ways in which to bring about the necessary movements of his extremities, with the recurrence of sufficient sensation to prevent the unconscious infliction of injury.

2. From the point of view of neurology, a patient is not considered cured until there has been reestablished to almost a normal degree the functions of sensation and motion in the region supplied by the affected nerve or nerves.

We have, in arriving at our conclusions, very closely followed the latter point of view, and consequently the improvements, marked improvement and cures are rather conservative estimates of the actual functional condition of the patients.

In practically all of the cases the date of injury anteceded the first examination, or operation, by at least five months, thereby giving a reasonable time for the appearance of some clinical signs of regeneration of the affected nerves.

The study of these cases shows that 511 nerves were affected up to the time this report was written. Of this number 259 were operated on, this number being exclusive of those who have not returned for a second examination. Two hundred and fifty-two were not operated on, or if operated on, have not returned for examination. Of the 252 nonoperative cases, 142 have been examined two or more times, 110 of the patients not having returned for a second examination. The actual results are therefore based on 400 injured peripheral

nerves which have been examined, with few exceptions, at varying intervals of from two to six weeks after the patients entered the hospital.

Of the 259 cases, forty-eight were neurolyzed, seventy-five were hersaged, and 136 were sutured. By neurolysis we mean that the nerve was freed from surrounding inflammatory tissue, and in a few cases the nerve sheath split. Hersage is the combing, or fiber disassociation, performed on a nerve by making a number of incisions with a very sharp knife in the longitudinal axis of the nerve. Suture is the approximation of two ends of a nerve after resection to normal fasciculi, and then with proper material sewing through the sheath, bringing the ends accurately together.

Table 1 shows the actual number of times that each nerve was involved. In our series of cases the ulnar nerve predominates, being affected 103 times; next in frequency comes the musculospiral, 85 times; then the median and sciatic, 75 and 74, respectively; then the external popliteal 37, and the brachial plexus, 26 times.

For purposes of study in this report we are going to eliminate the 110 nerves which have not had second examinations. Many of these patients are in the hospital; some of them have been operated on during the month of May, and will appear in later reports, thereby leaving 400 cases as a basis.

Of these 400 cases, operations were performed in 259, showing 65 per cent. of cases operative, and 35 per cent. nonoperative. Of this number 18 per cent. were neurolyzed, 28 per cent. were hersaged and 54 per cent. were sutured. I am rather inclined to believe that as these cases are observed for a longer period the percentage of operations will be somewhat increased.

Table 3 shows the actual number of nerves after neurolysis, hersage and suture that remained the same or were improved, much improved or cured. Of all operative cases, independent of the operation, about 50 per cent. remained the same; 35 per cent. were improved, 9 per cent. were much improved and 6 per cent. were cured. After neurolysis 20 per cent. remained the same; 45 per cent. were improved; 15 per cent. were much improved, and 20 per cent. were cured. After hersage 20 per cent. remained the same, 54 per cent. were improved, 16 per cent. were much improved, and 10 per cent. were cured. After suture 25 per cent. were improved, less than 1 per cent. were cured and the balance remained the same.

After neurolysis, more than 33 per cent. of the patients were markedly improved in from four to eight weeks; in less than twenty-four weeks nine were completely cured, some patients being cured as early as the fifth week. After hersage, at the end of twelve weeks,

25 per cent. of the patients were markedly improved, and in less than twenty-four weeks seven were cured. After suture, there were comparatively few patients that showed any improvement under sixteen weeks; after this time quite a number of them began to show improvement, especially in sensation; this is to be expected. There has been a marked improvement in the general appearance of the affected part, and in the comfort and mental attitude of the patients, and as time goes by we confidently anticipate a high percentage of cures in these cases.

#### PROGRESS IN OPERATIVE AND NONOPERATIVE CASES

In the operative cases, independent of operation, 44 per cent. of the cases were improved and 6 per cent. were cured; in the nonoperative cases 50 per cent. were improved and 17 per cent. were cured. If we exclude the suture cases, in which as yet it is too soon to expect much improvement, there remain 122 cases in which either herpage or neurolysis was performed. Of these cases twenty-six, or 21 per cent., remained the same; sixty-one, or 50 per cent., were improved; nineteen, or 16 per cent., were much improved, and sixteen, or 13 per cent., were cured. There were 142 cases in which no operation on the nerve was performed, and which were examined two or more times; in this group we find that forty-seven, or 33 per cent., remained the same; fifty-two, or 40 per cent., were improved; fifteen, or 10 per cent., were much improved, and twenty-eight, or 17 per cent., were cured.

On comparing the percentages in the two latter groups of cases, and taking into consideration the fact that the nonoperative cases were milder cases, and all gave a history of progressive clinical improvement, and that the operative cases were the more severe cases, and all gave a history of cessation of improvement for several months (and this in most cases was confirmed by two or more examinations), and that the large majority of these cases were examined from one to three months after operation, we find that 33 per cent. of the nonoperative cases remained the same as compared with 21 per cent. of the operative cases which remained the same; that 40 per cent. of the nonoperative cases were improved as compared with 49 per cent. of the operative cases; that 10 per cent. of the nonoperative cases were much improved as compared with 15 per cent. of the operative cases; and that 17 per cent. of the nonoperative cases were cured as compared with 13 per cent. of the operative cases.

These percentages, deduced from almost an equal number of operative and nonoperative cases in all of which the patients received their wounds in the same way, more or less about the same time, all subjected to similar early treatment and like treatment after arriving at the hospital, show first that operation in these cases, instead of doing

harm, hastens and makes more certain the improvement; in fact, we believe that if these 122 patients had not been operated on and had been treated in the same manner as the nonoperative cases, few, if any of them, would have shown very much improvement. A few patients were temporarily rendered worse after operation. This applies especially to the neurolyzed and hersaged cases; but almost without exception improvement followed and has continued. After hersage, in some cases improvement was delayed for a considerable time, and was then followed by a rather rapid disappearance of symptoms.

Disturbance of motion, sensation, pain and vasomotor and trophic manifestations did not become ameliorated or disappear simultaneously after the operation. In nearly all cases, pain was promptly relieved. In only a very few did pain persist, or occur in the distribution of the affected nerve after the operation. In no case of our series has the pain persisted for any length of time after operation. The vasomotor and trophic manifestations, in absence of vascular complications, in some cases were markedly improved, and in many cases disappeared after operations. There are, however a few in our series in which these disturbances have not been relieved by operation.

After neurolysis there is usually a prompt improvement in the sensory and motor disturbances.

After hersage there may be an improvement in motion or sensation, the two not necessarily improving, or returning at the same rate.

After suture the improvement that has been noted is return of the protopathic sensation, and progression toward the periphery of the point at which deep percussion tingling manifests itself.

*Brachial Plexus Injury.*—From a study of our tables, we find that there is a very marked tendency for injuries of the brachial plexus to be followed by a comparatively good functional restoration of the upper extremity, independent of whether they are operated on or not. We have had twenty-six cases of brachial plexus injury; of that number twenty-three have been examined two or more times; eleven were operated on, and twelve were not operated on. The operative cases all showed improvement, as did the nonoperative cases, although the manifestations of the operative cases were much more severe than of the nonoperative cases, and, according to the history, had ceased to improve for several months; however, following operation, improvement promptly occurred.

*Median Nerve Injury.*—The median nerve was affected in seventy-five instances; of that number fifty-nine were examined two or more times, forty-two were operated on, and seventeen were nonoperative. We noticed in this nerve that pain was rather a common symptom; vasomotor and trophic disturbances were frequently present, and when

the nerve was severed, the absolute loss of sensation was limited to the palmar surface of the two terminal phalanges of the index and middle fingers and dorsal surface of terminal phalanges of index and middle fingers; that on the palmar surface of the hand and proximal phalanges of the index and middle fingers, and the palmar surface of the thumb, tactile sensation was frequently lost, but pain stimulation gave rise to delayed, radiating, burning sensations. In those patients that were recovering, the muscles of the forearms supplied by the median nerve were the first to regain their function, with the possible exception of the flexor longus indicis. We notice in several cases that this muscle seemed to lag behind the other flexors in regaining its function. The thenar group of muscles was very slow in recovery, with the exception of the mild cases; tactile sensation was very long in reappearing in the terminal phalanges of the index and middle fingers.

*Musculospiral Nerve Injury.*—The musculospiral nerve, which was involved in eighty-five instances, operated on in thirty-seven cases, nonoperative in twenty-five cases and not returning for examination in twenty-three cases, shows a very marked tendency to recovery. This is evidenced in both operative and nonoperative cases. The sensory disturbances, even in instances of complete severance, are very limited, in most cases being limited to the dorsal surface of the proximal phalanx of the thumb and the small area on the dorsum of the hand at the base of the index and middle fingers. If this nerve is injured high up in the arm, or even in the axilla, the long head of the triceps may escape; when it is injured low down near the elbow, the entire triceps escapes. If the nerve is injured close to the elbow, the supinator longus is usually not involved. When the improvement takes place, the extensors of the wrist always recover before the extensors of the fingers.

*Ulnar Nerve Injury.*—The ulnar nerve was involved in 103 instances; of that number, sixty-six were operated on, eighteen were not operated on, and nineteen did not return for a second examination. Improvement in the ulnar nerve seems to be slower than in most of the other nerves; and, in fact, in those cases in which the nerve is very severely injured, although sensation and the function of the muscles of the forearm supplied by the ulnar nerve return in the expected time, the wasting and the power of the muscles of the hand supplied by this nerve does not seem to improve. In nearly all of our cases in which the ulnar nerve has been severely injured there has been very little improvement in the volume, tone and power of the muscles of the hand supplied by this nerve; in many cases, however, the functional result has been good.

We have noticed nothing very unusual in following our cases of sciatic, external or internal popliteal injuries, with the possible exception of the facts that vasomotor and trophic disturbances are more apt to occur in the distribution of the external popliteal than the internal popliteal; that these disturbances rather disappeared after operation; that recovery in the domain of the external popliteal is usually more complete than that in the internal popliteal; that in injuries of the sciatic nerve, the fasciculi which go to the external popliteal are more apt to be injured than those that go to the internal popliteal, and that in those cases in which causalgia occurs following injury of the sciatic nerve, the pain usually occurs in the distribution of the internal popliteal.

#### CONCLUSIONS

1. Careful neurological examinations should be made of each patient. When this examination justifies the diagnosis of complete physiologic block, taken with a history of cessation of improvement for from four to eight weeks, operation is indicated.

2. The presence of persistent pain or marked vasomotor and secretory disturbances, with a history of cessation of improvement for some time, calls for operative interference.

3. Following operation as soon as the surgical condition permits, energetic physiotherapy should be instituted; this should consist of passive massage; active exercise; electrotherapy, the current which will produce muscular contraction being used; proper splinting to prevent deformity; overstretching of paralyzed muscles, or contraction of paralyzed muscles, while still permitting as much freedom in motion of the affected part as possible.

4. The patient should be encouraged to cultivate a tranquil, optimistic attitude toward his disability, and to cooperate in his treatment.

5. From the neurological examinations, supported by subsequent operative findings, it is our opinion that from 60 to 75 per cent. of the patients at this hospital who have nerve injuries would not recover lost function without operative interference.

6. The operative cases have not been under observation a sufficient length of time to arrive at a more than tentative conclusion as to ultimate outcome.

7. The neurolysis and hersage cases have shown a considerable number of improvements, which we believe will be increased at a later date. Although measurable, objective improvement in the suture cases has been very limited, it is yet too early to expect any other result.

## BULBAR PARALYSIS OR AMYOTROPHIC LATERAL SCLEROSIS?

A CLINICO-PATHOLOGIC NOTE \*

G. B. HASSIN, M.D.

Attending Neurologist, Cook County Hospital

CHICAGO

After Duchenne's first description of progressive bulbar paralysis<sup>1</sup> this disease was generally looked on as an independent clinical and pathologic entity. Later, numerous observations showed that it may form a part of the clinical pictures of amyotrophic lateral sclerosis, of chronic anterior poliomyelitis (subacute general anterior spinal paralysis of Duchenne) and progressive muscular atrophy (type Aran-Duchenne). The symptoms and signs are sometimes so confusing that a differential diagnosis between these four types is very difficult, and some authors (Starr,<sup>2</sup> Gowers<sup>3</sup>) have come to look on at least some of these conditions as practically identical diseases. The following clinico-pathologic report tends to demonstrate the identity of amyotrophic lateral sclerosis and chronic bulbar paralysis.

### REPORT OF A CASE

Mrs. E. R., 62 years old, admitted on Dec. 13, 1918, to the county hospital, service of Dr. Frederick Tice, complaining of difficulty in swallowing, talking and walking, about one and a half years previously had noticed weakness of the upper and lower extremities and swelling of the latter. The weakness progressed and at the time of admission she could neither walk without support nor pick up small objects. During the past year her voice had become hoarse, "stumbling," her speech unintelligible, and for the last two months she had had dysphagia, especially for liquids.

*Previous History.*—She had had asthma since childhood, diphtheria at the age of 8. Syphilis and other constitutional diseases were denied. She had amenorrhea at the age of 36.

*Family History.*—Her father and mother died of old age.

*Examination.*—The patient sat upright in bed with the head well forward and her back markedly bowed anteriorly, constantly coughing and with a

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\* From the pathology laboratories of Cook County and Psychopathic Hospitals, Chicago.

1. Duchenne (de Boulogne): Paralyse progressive de la langue, du voile du palais et des lèvres, Arch. gén. de méd. 2:283, 431, 1860.

2. Starr, M. A.: Organic and Functional Nervous Diseases, Ed. 3, Philadelphia, Lea & Febiger, 1909, p. 280.

3. Gowers, W. R.: Diseases of the Nervous System, Philadelphia, P. Blakiston's Son & Co., 1892, Vol. 1.

peculiar facial expression. The muscles of the lower half of the face were drawn, retracting the corners of the mouth which was slightly open, the frontal muscles contracted, saliva constantly drooling. She could wrinkle the forehead, close the eyes and move them in every direction. The muscles of mastication and of lips, chin and tongue were markedly atrophied. The tongue could not be protruded beyond the teeth; it was thin, flabby, corrugated and showed marked fibrillary twitchings. The uvula and soft palate were motionless and did not respond to irritations; they appeared atrophied. The neck was thin, not rigid, the shoulders high, held as in forced breathing. The lungs revealed numerous high-pitched piping sounds typical of asthma. The heart was slightly enlarged, but showed no murmurs. The abdomen was negative.

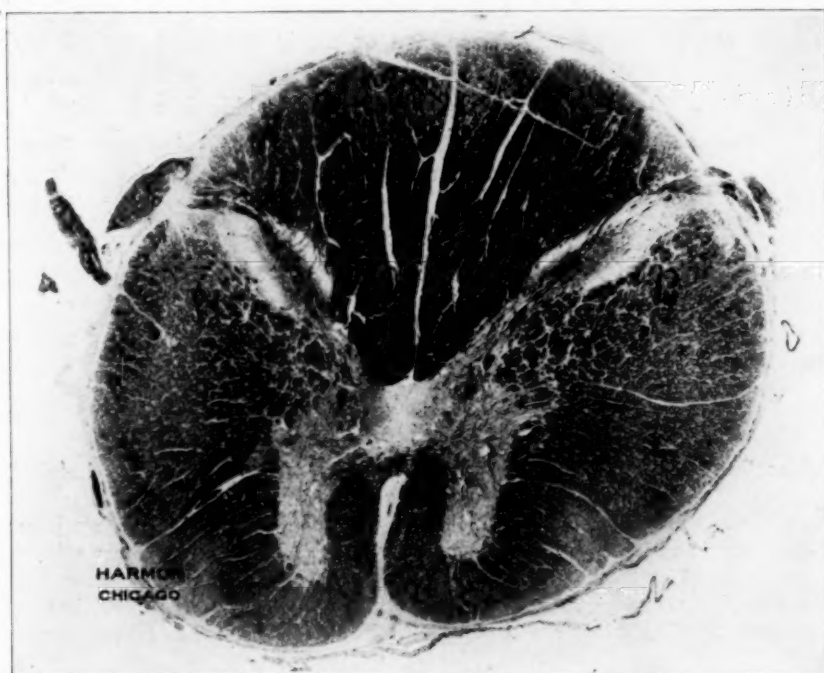


Fig. 1.—Second cervical segment. Posterior roots cross the posterior horns and are well developed, in contrast to the anterior horns, which appear smaller than normal and without any anterior root fibers visible. Fibers of the eleventh nerve, normally crossing the lateral columns, are also absent. The posterior, the lateral and anterior columns, including the pyramidal tracts (Py), do not show any visible or marked changes. Weigert-Pal stain,  $\times 9$ .

*Extremities.*—Active and passive movements were possible, without rigidity, spasticity or deformities. The feet were drooping; small muscles of the hands, the biceps, deltoid, and calf muscles were somewhat atrophied, especially the hand muscles, and fibrillary twitchings were quite marked in the deltoid and biceps of both sides. Reflexes: Pupillary, normal; triceps, knee, ankle and jaw jerks were all exaggerated, especially on the right; no clonus. Oppenheim's sign and the Chaddock and Babinski reflexes were absent. The voice was hoarse,

hardly audible; speech unintelligible, anarthric; mentality, good. Urine contained albumin, hyaline and granular casts. No serologic tests were recorded.

The anarthria, dysphagia and respiratory difficulties progressed and on Jan. 10, 1919, the patient died.

*Necropsy Examination.*—The necropsy three days later, revealed emphysema and edema of the lungs, hypostatic hyperemia of the liver, diffuse nephritis, disseminated petechial hemorrhages in the gastric mucosa, moderate senile sclerosis of the aorta, marked atrophy of the interossei muscles of both hands, extensive fibrous peritonitis; the cerebral pia congested, transparent, not thickened and not adherent to the brain; normal sulci, convolutions and ventricles; the seventh, ninth and twelfth cranial nerves were visibly atrophied.

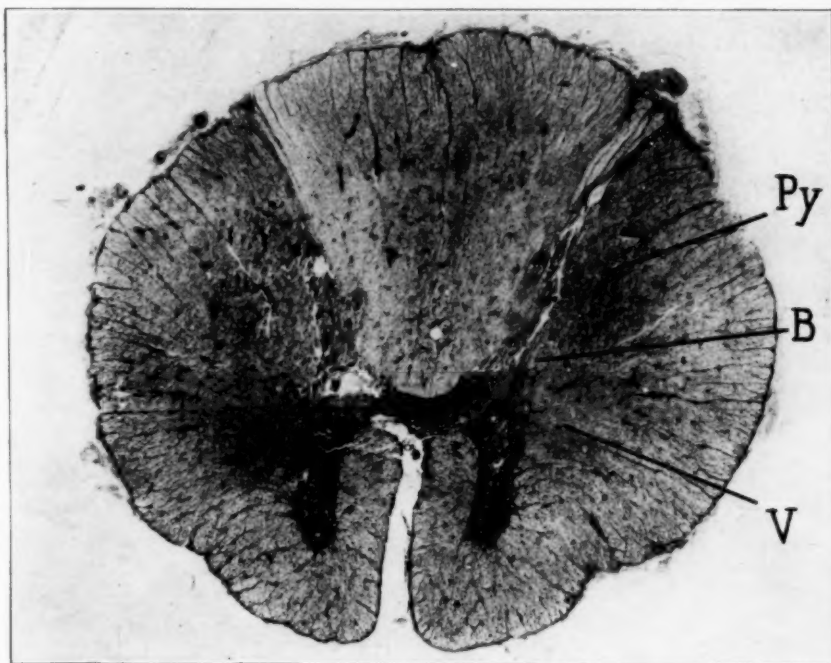


Fig. 2.—Tenth dorsal segment. The posterior and anterior columns are normal, showing, especially the posterior columns, a great number of vessels. In the lateral columns, the tracts of Flechsig and Gowers are also normal, while the areas of the crossed pyramidal tracts and the limiting lateral layer, its ventral portion, are degenerated, appearing darker stained than the normal portions of the spinal cord. *Py*, crossed pyramidal tracts; *B*, dorsal part of the lateral limiting layer, known as Bechterew's medial bundle of the lateral columns, is not degenerated; *V*, ventral part of the lateral limiting layer shows marked degeneration. Alzheimer-Mann stain; frozen section;  $\times 13$ .

Sections of the medulla, cervical and upper portions of the dorsal cord stained with Weigert-Pal showed atrophy of the nuclei of the twelfth, ninth, tenth and seventh cranial nerves and the fifth motor nerve and the corresponding nerve roots with practically normal white tracts in medulla and cord. The pyramidal fibers, as the photomicrograph (Fig. 1) shows, were normal in

amount though they appear less stained than those of the posterior columns. Both white and gray matter showed numerous congested, thickened and slightly infiltrated vessels (Figs. 2 and 3). The same excessive vascularization was present in the pia which was markedly infiltrated with lymphocytes (Fig. 3). Longitudinal sections of the pyramidal tract, stained with Mallory-Jacob (fuchsin and anilin blue), Alzheimer-Mann (methyl-blue eosin), and, still better, with Marchi or Bielchowsky and counterstained with the former methods

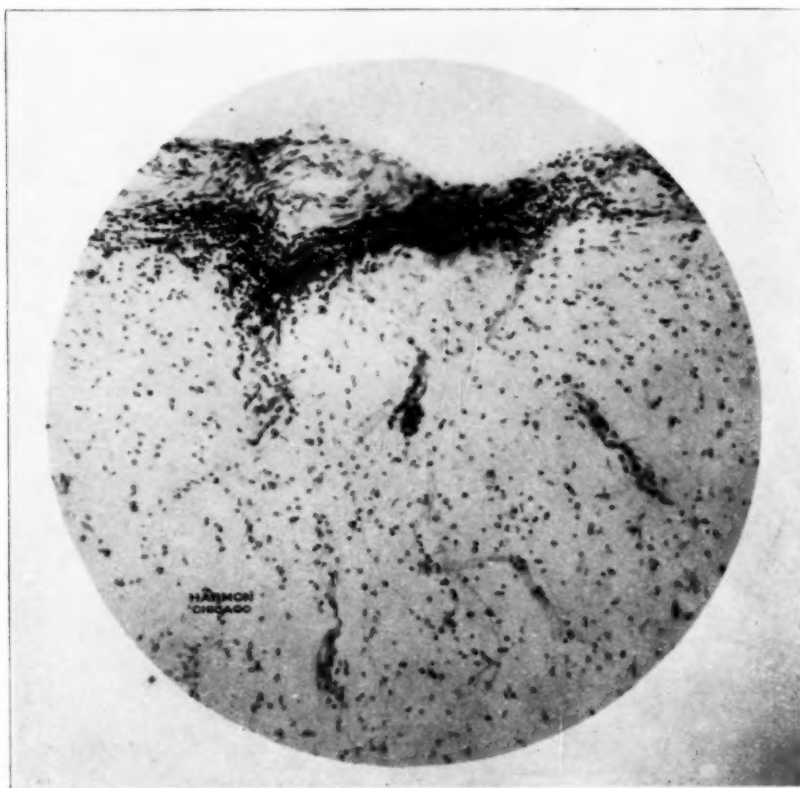


Fig. 3.—Infiltration of pia and vessels of spinal cord. The vascular infiltration is mild. Toluidin blue stain;  $\times 110$ .

revealed quite an unexpected amount of changes which briefly can be summed up as follows (Fig. 4):

1. There were numerous nerve fibers with perfectly normal myelin sheath and axon.
2. A number of fibers were deprived of myelin and consisted only of tortuous and slightly swollen axons.
3. Fragments of myelin, so-called Marchi globules, lodged within larger or smaller vacuoles surrounded by glia fibers.

4. Vacuoles filled with gliogenous formations described by Jacob<sup>4</sup> as myeloclasts and myelophages.

5. There was an abundance of glia fibers covered with glia nuclei and large protoplasmic glia cells with one or two nuclei frequently containing fat droplets and many small processes.

6. There were gitter cells of various types as described by Jacob, some containing a large vacuole with fragments of myelin or axon (gitter cells- $\alpha$ );

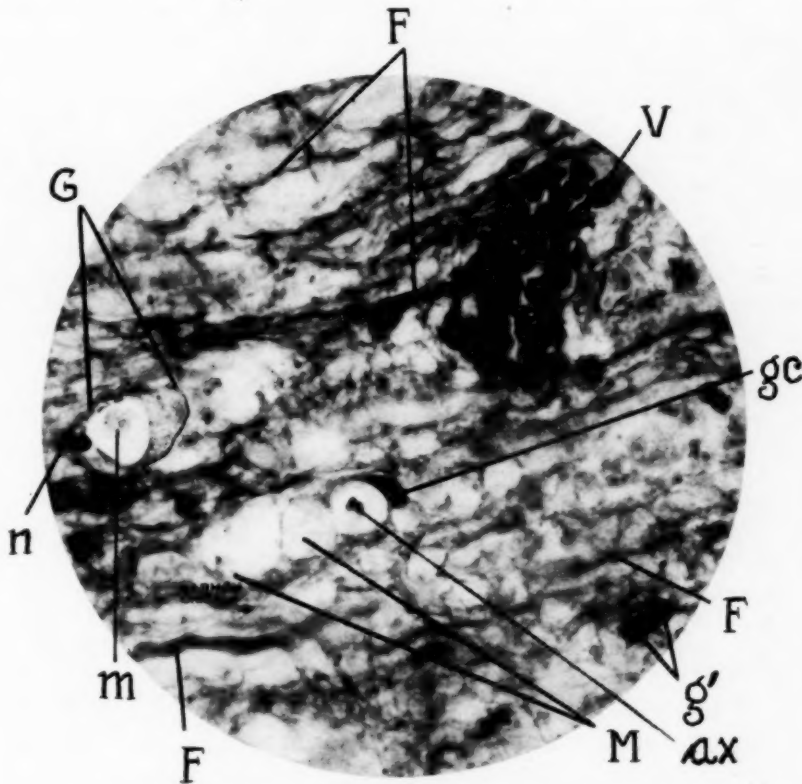


Fig. 4.—Longitudinal section of the lateral pyramid of the spinal cord. Normal fibers ( $F$ ) are separated by a great number of vacuoles, some of which contain remnants of nerve fibers (myelin and axons);  $F$ ,  $F$ , nerve fibers;  $V$ , vessel;  $G$ , gitter cell- $\alpha$ ; its right half is like a typical gitter cell where with the help of a lens one can see its vacuolated structure, the left half is represented by a vacuole containing a fragment of myelin ( $m$ );  $n$ , its nucleus;  $gc$ , glia cell with a dark nucleus and processes enveloping a fragment of axon ( $ax$ );  $g'$ , glia nuclei;  $M$ , myelin globules (Marchi globules). Alzheimer-Mann stain;  $\times 600$ .

4. Jacob, A.: Ueber die feinere Histologie der secundären Faser-degeneration in der weissen Substanz des Rückenmarks, Nissl-Alzheimer's Arbeiten 5:182, 1912.

these cells were the most numerous; other gitter cells showed some large processes (gitter cells- $\beta$ ) and very few typical gitter cells round in shape without any processes and densely packed with fat globules (gitter cells- $\gamma$ ).

7. Numerous vacuoles devoid of contents giving the section an areolar sieve-like appearance were found.

Of the outlined changes the so-called Marchi globules were the most frequent, indicating the degenerative process to be in the earliest stages. The same changes, but much less pronounced, could be found in the pyramids of the medulla, while the posterior columns, the spino-cerebellar tracts and various regions of the medulla showed no changes at all, except excessive vascularization and dense glia formation around the vessels.

Especially interesting were changes in the cortical motor area, as well as in the gray matter of the medulla and spinal cord. The latter revealed scarcity and atrophy of the anterior horn ganglion cells, abundance of glia fibers and glia nuclei, some amyloid bodies and excessive vascularization; in short, changes analogous to those in amyotrophic lateral sclerosis. The nuclei of the medulla (the twelfth, tenth and ninth were principally studied) showed a number of cells more or less preserved, but as a rule there was a great lack of ganglion cells. The striking feature was abundance of vessels in the nuclei (Fig. 5) which were literally studded with small new-formed capillaries, some vessels having been congested, but not infiltrated. The same abundance of vessels was seen in the calamus scriptorius, formatio reticularis, nucleus ambiguus and olivary bodies. The elastica stain of Weigert shows especially well the extraordinary number of vessels even as far as the corpora quadrigemina and cerebral cortex. The motor area of the latter, in addition, exhibited marked structural changes in some cells, though many ganglion cells, especially of the upper layers, appeared quite normal. Many cells of the deeper strata (fourth to sixth) were uniform in their staining, granular, broken up, with a pale dislocated nucleus, sometimes invaded by glia cells, etc.; in short, there were neuronophagia, chromatolysis, cell-shrinking, satellitosis which probably accounted for the described condition in the pyramidal tracts.

#### COMPARISON OF AUTHOR'S CASE WITH BULBAR PARALYSIS AND AMYOTROPHIC LATERAL SCLEROSIS

This combination of changes in pyramidal tracts and anterior horn cells certainly justifies a diagnosis of amyotrophic lateral sclerosis. Yet, when compared with changes as described by me<sup>5</sup> in a typical case of amyotrophic lateral sclerosis, there could be pointed out some differences. Thus, in amyotrophic lateral sclerosis the pyramidal fibers in the lateral, and partly in the anterior, columns were densely packed with fat-like (lipoid) globules, in Herxheimer (scarlet red) and Marchi specimens (Fig. 6), while in the present case such changes were few and slight. The degeneration evidently did not progress far enough to furnish products that would stain with osmic acid or scarlet

5. Hassin, G. B.: Histopathological Changes in a Case of Amyotrophic Lateral Sclerosis, *Med. Rec.* 91:228 (Feb. 10) 1917.

red, but only such small fragments as could be demonstrated with Alzheimer-Mann, or Mallory-Jacob stains. In typical amyotrophic lateral sclerosis the glia changes are much more striking, resembling those found in various stages of experimental secondary degeneration so well described by Jacob. On the other hand, vascular changes, especially in the affected nuclei, are missing or at least not recorded in amyotrophic lateral sclerosis. Their presence in bulbar paralysis has been pointed out by Duchenne and Joffroy,<sup>6</sup> and Eisenlohr.<sup>7</sup> In amy-

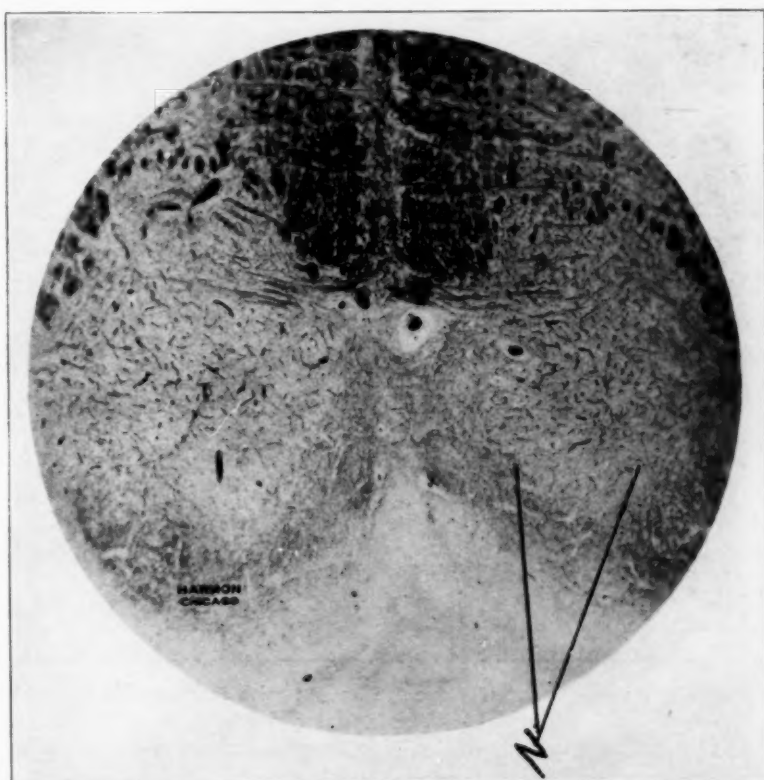


Fig. 5.—The nuclei of the twelfth nerve, separated in the front by the medial fillet. The anterior three fourths of the nuclei exhibit an abundance of vessels; the posterior fourth (N) is quite normal, appearing pale and arched. Weigert-Pal stain;  $\times 27$ .

otrophic lateral sclerosis many vessels show infiltration of their adventitial spaces with fat which I could not find in this case, in which,

6. Duchenne and Joffroy: De l'atrophie aiguë et chronique des cellules nerveuses de la moelle et du bulbe rachidien, *Arch. de physiol.* **3**:497, 1870.

7. Eisenlohr: Klinische und Anatomische Beiträge zur progressiven Bulbärparalyse, *Ztschr. f. klin. Med.* **1**:435, 1880.

however, the vessels, especially of the spinal cord and cortex were markedly infiltrated with lymphocytes. The pia in the present case also appeared infiltrated, exclusively with lymphocytes, which is not the case in amyotrophic lateral sclerosis. The foregoing differences, however, are but slight, probably accidental (like, for instance, the pial infiltration), being quantitative rather than qualitative, and a diagnosis of amyotrophic lateral sclerosis in the present case safely could be made. The outlined pathologic changes thus afford additional proof

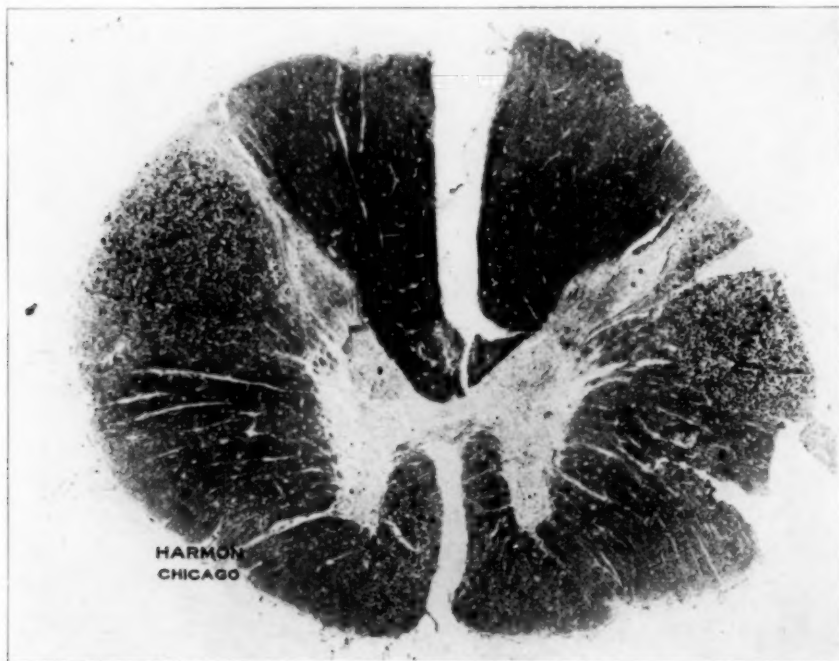


Fig. 6.—Tenth dorsal segment from a patient with amyotrophic lateral sclerosis. The lateral and somewhat the anterior pyramids are converted into a mass of drops and droplets of fat. No fat globules are to be found in the posterior columns, Gowers' or Flechsig's tracts, or in the lateral limiting layer of either side. A frozen section photographed four years after it was stained with Herxheimer scarlet red stain. A hand lens will bring out the details better;  $\times 15$ .

that bulbar paralysis is practically nothing but amyotrophic lateral sclerosis. Carefully studied cases of chronic bulbar paralysis without concomitant pyramidal tract involvement are practically unknown. The few cases reported (Charcot,<sup>8</sup> Duchenne and Joffroy,<sup>6</sup> Leyden,<sup>9</sup>

8. Charcot, J.: Note sur un cas de paralysie glosso-laryngée, *Arch. de physiol. norm. et path.* **3**:247, 1870.

9. Leyden, E.: Progressive Bulbärparalyse, *Arch. f. Psychiat.* **2**:429, 643, 1870.

Eisenlohr<sup>7</sup>) in which the lateral columns were found intact are the earliest recorded, and it is probable, as Leyden himself admits, that possibly in these cases the pyramidal changes were overlooked. Amyotrophic lateral sclerosis may begin and run its course as typical bulbar paralysis (Hun,<sup>10</sup> Maier,<sup>11</sup> Kussmaul,<sup>12</sup> Leyden,<sup>13</sup> Dejerine<sup>14</sup>), though a majority of cases recorded show that it *follows* the lateral column lesion. In our case, bulbar and pyramidal lesions apparently occurred simultaneously. The simultaneous combined lesion of the motor area of the brain, of the motor nuclei of the bulb and spinal cord will give a clinical picture of amyotrophic lateral sclerosis.<sup>5</sup> But it may happen that the medullary nuclei will be *principally*, though *not exclusively*, involved. Then Duchenne's picture of glosso-labio-laryngeal paralysis will obtain. Preponderating involvement of anterior horn cells of the spinal cord will result in progressive muscular atrophy, or subacute general anterior spinal paralysis of Duchenne, also known as chronic anterior poliomyelitis. Finally, it may happen that the cortical motor cells will be principally affected resulting in a peculiar syndrome described as progressive hemiplegia, triplegia, quadriplegia, etc. Whatever the variety, probably in each case there will be found motor cell changes in the cortex, in the medulla oblongata, pons and spinal cord. The present case demonstrates this combination in bulbar paralysis, it being also present in amyotrophic lateral sclerosis, and Alzheimer<sup>15</sup> showed the same thing in progressive muscular atrophy. Whether the same applies in progressive hemiplegia remains to be seen, as there are no exhaustive histopathological records of this syndrome.<sup>16</sup> Here it must be pointed out that our knowledge of the histopathology of bulbar paralysis, amyotrophic lateral sclerosis, progressive muscular atrophy and progressive hemiplegia is yet in a very

10. Hun, E. R.: Labio-glosso-laryngeal Paralysis, *Am. J. Insan.* **28**:194, 1871.

11. Maier, R.: Ein Fall von vorschreitender Bulbärparalyse, *Virchow's Arch. f. Path. Anat.* **8**:641, 1878.

12. Kussmaul, S.: Ueber fortschreitende Bulbärparalyse und ihre Verhältnisse zur progressiven Muskelatrophie, *Samml. klin. Vortr. (Volkmann's)* **1**:439, No. 54, *Innere Medizin*.

13. Leyden, E.: Ueber progressive amyotrophische Bulbärparalyse und ihre Beziehungen zur symmetrischen Seitenstrangsklerose, *Arch. f. Psychiat.* **8**:641, 1878.

14. Dejerine, J.: Etude anatomique et clinique sur la paralysie labio-glosso-larynge, *Arch. de physiol. norm. et path.* **152**:180, 1883.

15. Alzheimer, A.: Ueber einen Fall von spinaler progressiver Muskelatrophie mit hinzutretender Erkrankung bulbärer Kerne und der Rinde, *Arch. f. Psychiat.* **23**:459, 1892.

16. Hassin, G. B.: A Case of Amyotrophic Lateral Sclerosis in the Form of Progressively Developing Triplegia, *New York M. J.* **106**:932 (Nov. 17) 1917.

unsatisfactory state, contributions to these problems are not only very sparse, but very incomplete. Thus, in the majority of them, there is no record of the changes in the brain or other portions of the central nervous system, which, if studied at all, were followed up with the old methods, while the condition of the glia was not fully described. Further studies with the newer methods are absolutely essential for establishing the real relationship of these four syndromes which clinically differ, in my opinion, by the localization of the parts *principally*, but not *exclusively* involved.

31 North State Street.

## LETHARGIC ENCEPHALITIS \*

JOSEPHINE B. NEAL, M.D.

NEW YORK

For the past nine years we have had at the research laboratory of the health department of New York City a meningitis division, the function of the members of which is to see in consultation all kinds of meningeal conditions for differential diagnosis and treatment. In this connection we have seen over 1,000 cases of meningitis of various kinds, 600 or more cases of poliomyelitis and over 700 cases of meningism with various diseases, besides small numbers of cases of numerous other conditions, so that we have had a fairly good background for the study of a new type of meningeal or cerebral disease.

Last October, as a member of the research laboratory of the health department of New York City, I began to see a new type of disease. The majority of these patients gave a history of influenza followed in a varying length of time by headache, drowsiness and apathy, usually accompanied by a low irregular fever, strongly suggesting a slowly developing tuberculous meningitis. One of the earliest patients, "X," showed extreme restlessness instead of drowsiness, marked muscular weakness and some paralysis of the cranial nerves. These conditions suggested a variety of diagnoses. Meningism seemed possible in certain instances, perhaps caused by some gastro-intestinal disorder, or it may have followed, instead of accompanied, influenza; this, however, was ruled out as the spinal fluid showed a marked increase in the protein elements and cells instead of being normal, as is the rule in meningism. Syphilitic disease was suspected, especially in "X," but this was disproved by the negative Wassermann test and by the character of the gold chlorid curve. Brain tumor must be differentiated in certain of the more severe cases, and this differentiation has proved a stumbling block to some very eminent neurologists. Tuberculous meningitis was considered in many cases, but this diagnosis was discarded because of failure to find the tubercle bacillus either by smear or by animal inoculation, the normal reduction of Fehling's solution and the favorable termination. In some instances the encephalitic type of poliomyelitis was suggested by the clinical picture and by the spinal fluid findings, but this type is rare even in epidemics of poliomyelitis,

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\* From the Research Laboratory Department of Health, New York City.

TABLE 1.—SALIENT FEATURES OF FORTY CASES OF LETHARGIC ENCEPHALITIS

Case No.	Age	History of Influenza	Type of Onset	Lethargy	Asthenia	Headache	Pupils	Reflexes	Temperature	Vomiting	Miscellaneous	Duration	Outcome	Remarks
14	12 wk. M.	+	Slow	Slight	.....	.....	—	—	100	+	.....	7 days	Recovered	
388	8 mo. M.	+	Slow	+	.....	.....	Int. strabis.	—	To 105	—	.....	2 weeks	Died	
398	9 mo. F.	+	Sudden	+	.....	.....	.....	—	Slight	—	.....	3 weeks	Recovered	
32	15 mo. M.	+	Slow	+	.....	.....	.....	—	.....	+	.....		Died	
5	15 mo. M.	+	Slow	+	.....	.....	—	?	+	+	.....	4-5 weeks	Recovered	
9	18 mo. F.	+	Slow	+	.....	.....	Int. strabis.	N.	102-103	+	.....	1 week	Died	
371	20 mo. F.	+	Slow	+	.....	.....	Int. strabis.	—	Slight	+	.....	3 months	Died	
94	2 yrs. M.	+	Slow	+	.....	.....	—	N.	101	+	.....	10 days	Recovered	
49	2 yrs. M.	+	Slow	+	.....	.....	Rt. facial	—	104	+	.....	6 days	Died	
54	3 yrs. M.	+	Sudden	+	.....	.....	—	—	104	+	.....	4 weeks	Recovered	
16	3 yrs. M.	+	Slow	+	.....	.....	.....	—	Slight	+	.....	3 weeks	Recovered	
87	3½ yrs. M.	+	Sudden	+	.....	.....	.....	—	100	+	.....	10 days	Recovered	
351	4 yrs. F.	Probable	Sudden	+	.....	.....	.....	—	Slight	+	.....	Less than 1 month	Recovered	
137	4 yrs. M.	—	Sudden	+	.....	.....	.....	—	Slight	+	.....	2 weeks	Recovered	
391	4½ yrs. F.	—	Slow	+	.....	.....	.....	N.	100.5	—	.....	2 weeks	Recovered	
395	7 yrs. F.	—	Slow	+	.....	.....	.....	+	101.5	—	.....	2-3 weeks	Recovered	
19	7 yrs. M.	+	?	+	.....	.....	.....	—	104	—	.....	3 weeks	Died	
41	7 yrs. M.	+	Slow	+	.....	.....	.....	—	104	—	.....	4 weeks	Recovered	
80	9 yrs. M.	—	Sudden	+	.....	.....	.....	N. rt.	Slight	—	.....	2 weeks	Recovering	
357	9 yrs. M.	—	Slow	+	.....	.....	Facial Slight dysphagia	—	104	—	.....	1 month	Recovered	
46	9 yrs. M.	—	Slow	+	.....	.....	.....	++	+	—	.....	3 months	Improving	
25	10 yrs. M.	+	Sudden	+	.....	.....	.....	—	+	+	.....	2 weeks	Recovering	
109	10 yrs. M.	+	Slow	+	.....	.....	.....	N.	Slight	+	.....	7-8 days	Died	
38	11 yrs. M.	+	Slow	+	.....	.....	Rt. facial	D.	101	—	.....	2 months	Recovered	Mental examination showed it had been abnormal some years, not sequel of encephalitis

331	14 yrs. M.	?	Sudden	+	+	+	+	+	+	102	—	.....	Delirium.....	3 weeks	Recovered	
332	16 yrs. M.	—	Sudden	+	+	+	+	+	+	To 106.6	—	.....	.....	3 days	Died	
Y	18 yrs. M.	—	Sudden	++	++	++	++	++	++	102	—	.....	Slight delirium at times.....	3-4 weeks	Recovered	
24	20 yrs. M.	+	Slow	+	+	+	+	+	+	103	—	.....	Paralysis after 1 week, delirium	1 month	Recovered	
48	20 yrs. M.	—	Sudden	+++	+++	+++	+++	+++	+++	103	—	.....	Leg spastic, marked sweating, mask-like expression, tremors, delirium	1 week	Died	
333	23 yrs. M.	+	Slow	++	++	++	++	++	++	+	+	.....	.....	1 month	Recovered	
333	25 yrs. ?F.	+	Slow	+++	+++	+++	+++	+++	+++	103	.....	.....	Pregnant, normal delivery, delirium at times	Over 2 months	Recovered	
66	30 yrs. ?M.	+	Slow	+++	+++	+++	+++	+++	+++	90-103	++	.....	Restlessness, marked tremors, mask-like expression	6 weeks	Recovered	
103	30 yrs. M.	+	Slow	+	+	+	+	+	+	—	N.	.....	Disturbance of vision, vacant expression, delirium at times	?	Recovering	
27	32 yrs. M.	+	Slow	+++	+++	+++	+++	+++	+++	102	D.	.....	Nystagmus, dizziness, diplopia, tremors, mask-like expression, spasticity	4 months	Recovered	
50	35 yrs. M.	—	Slow	++	++	++	++	++	++	101-102	++	.....	Diplopia, difficult speech, dizziness	2 months	Recovering	Mental depression, slight paralysis
72	43 yrs. M.	+	Sudden	++	++	++	++	++	++	—	N. left D. rt.	.....	Dizziness, diminished sensitivity on right upper and lower extremities	2 weeks	Recovering	Slight weakness
332	45 yrs. M.	+	Slow	++	++	++	++	++	++	103	++	.....	B a b i n s k i incontinence, sweating, mask-like expression, delirium	4 months	Recovered	Rapid pulse
68	50 yrs. M.	—	Slow	+++	+++	+++	+++	+++	+++	Slight	D.	.....	Babinski, mask-like expression	3 months	Recovered	
X	50 yrs. ?M.	+	Slow	—	+	+	+	+	+	+	—	.....	Irritability, pain, restlessness	3 mo.; facial paralysis after 5 mo., nearly gone at 6	Practically recovered	Slight paralysis remaining
52	50 yrs. M.	—	Sudden	++	++	++	++	++	++	Slight	N.	.....	.....	1 month	Died	

and during the fall and winter the number of cases of even the spinai type has been extremely small. Finally, I began calling these cases influenzal encephalitis, since the symptoms were those of encephalitis and since they so often followed influenza. Not until midwinter did I designate these cases lethargic encephalitis, as it was not until that time that I encountered patients with lethargy, asthenia and oculomotor palsies, the characteristic triad of symptoms described by the English and French. Table 1 gives some of the salient features of forty cases in which the diagnosis seems to be well established. A study of the table shows: (1) wide age distribution—from twelve weeks to over fifty years. From the nature of our work we see an unusually high proportion of children. (2) The large proportion of males, thirty-three out of forty. Tucker reports nine males out of eleven cases. In other reports, usually of smaller numbers, there has not been this marked difference. (3) The history of an attack, clinically influenza, in twenty-seven of the forty cases. It will be noted that the onset is more often slow than sudden, and that lethargy and asthenia are nearly constant symptoms, while cranial nerve or other palsies are present in less than half of the cases. It is possible that our list includes a rather high proportion of mild cases, since we are called in consultation where an early tuberculous meningitis is suspected. Where lumbar puncture is not so freely resorted to, I fancy that many of these milder cases are unrecognized.

#### REPORT OF CASES

Certain patients are perhaps of sufficient interest to deserve special attention.

CASE 1.—Patient 357 had a typical mild case. He had influenza early in October. About the middle of October he became gradually worse, with headache, vomiting, constipation (which the French call the meningitic triad), irregular fever (102 to 104 F.), and marked apathy. This history was very suggestive of tuberculous meningitis. I saw him October 27. He was then somewhat improved. His mental condition was nearly normal, there was no well-marked stiffness of the back, but he still had fever, his patellar reflexes could not be obtained, and there was a moderate Kernig's and Macewen's sign. Lumbar puncture revealed clear fluid under pressure showing only a slight increase in the proteid contents. The case cleared up quite promptly.

CASE 2.—Patient 66 was a physician about 30 years of age. He had an attack of influenza which began February 18 and lasted for six or seven days. While there was general improvement after the attack, he continued to suffer with severe headache, and late in February he began to have fever, which ran as high as 103 F., and marked apathy. I saw him March 12. His face was entirely expressionless and he made no response whatever when requested to smile; apparently there was great weakness of the facial muscles on both sides. At times there was ptosis. He was unable to move, but frequently asked to be turned as he was uncomfortable if left long in one position as a tremor involving all the limbs was then most likely to develop. His pupils

were equal and reacted to light; the knee jerks were slightly increased and he was entirely clear mentally and very anxious about his condition. As Dr. Foster Kennedy very aptly expresses it, these patients are emotionally stuporous and intellectually bright. About 35 c.c. of clear spinal fluid was withdrawn, showing great increase in cells and proteid contents; a negative Wassermann test was made. He was able to be out of bed by the end of March and after another month had quite recovered, though he was still a little weak and suffered slightly from insomnia.

CASE 3.—Patient 393, five months pregnant, had an attack of influenza two weeks before the onset of the encephalitis, which began gradually early in December with headache, chill and fever, vomiting, sweating and delirium. I saw her December 14, at which time she was stuporous. There was some stiffness of the neck, and a right facial paralysis. About 25 c.c. of clear spinal fluid were withdrawn, which showed great increase in cells and in the protein elements, and a negative Wassermann test. A guinea-pig inoculated with the fluid gave a negative reaction for tuberculosis. Her condition remained the same for two weeks or more and then she gradually recovered. The facial paralysis cleared up, and she had a normal delivery at term.

CASE 4.—Patient 38, a boy of 11 years, was seen February 4. Late in January he had what had been diagnosed as a mild attack of influenza. On the 31st he began to have gradually increasing headache, apathy, and low fever. When examined, February 4, he was stuporous, the pulse rapid (120), and quite arrhythmic. His temperature was between 100 and 101 F. The right pupil reacted sluggishly to light, the left was normal. The right patellar reflex was increased, and there was a right facial paralysis. Fifteen c.c. of spinal fluid withdrawn on this occasion and 25 c.c. withdrawn two days later showed slight increase in cells, albumin and globulin. The Wassermann test was negative. The patellar reflexes became equal in a few days. By the 11th he was greatly improved. The stuporous condition had disappeared, and the pulse, though still rapid, had ceased to be arrhythmic. Later, however, he became mentally disturbed and was violent. By the middle of April, he seemed to have recovered, but his mother reported that he had a voracious appetite and was very troublesome and difficult to manage. At first it seemed that his condition was due to the encephalitis but an investigation of the case revealed the fact that he had been in classes for atypical children for two or three years. He was examined by Dr. Coffin of the department of education for New York City, and was found to belong to the hyperactive, precocious type of children that it is so difficult to deal with satisfactorily.

CASE 5.—Patient 46, a boy of 9 years, is of interest on account of the long duration of his disease. He was admitted to Willard Parker Hospital with a history of being ill for three days. He was then comatose, had slight rigidity of the neck and Kernig's sign, exaggerated knee jerks, positive Brudzinski sign and Babinski reflex. He ran an irregular temperature, from 100 to 103 F. until February 19, after which it was below 100 until March 14. At times his pulse and respiration were irregular. He became progressively worse and by February 20 had marked rigidity of the entire body, and a mask-like, expressionless face. He became unable or unwilling to swallow and had to be tube fed. The white blood count was 15,000, 81 per cent. polymorphonuclears. The first lumbar puncture, February 13, showed a clear fluid with moderate increase in cells, 60 per cent. polymorphonuclears, moderate increase in protein elements, normal reduction in Fehling's sign and negative Wassermann test. Fluid withdrawn February 14, was slightly blood-tinged so that it had a

somewhat hazy appearance. Therefore, with the clinical picture resembling meningitis so strongly, and an excess of polymorphonuclears in the first fluid, serum was given. Of course, this obscured the spinal fluid picture for some time. During all this time the child did not speak and has not up to the present time. There were frequent muscular twitchings. Early in March he began to move his head and a little later his legs and arms, and seemed to be progressing toward recovery. On March 14, however, the temperature rose to 106 F., he perspired profusely and seemed to be in a desperate condition. A blood culture at this time was negative. Twenty c.c. of spinal fluid were withdrawn under some pressure, showing a moderate increase in cells, 80 per cent. mononuclears, increase in albumin and globulin, and a normal reduction of Fehling's. On March 15 he began to improve; his temperature dropped to 100 F. Since that time his condition has shown some improvement, and he has gained in weight. A peculiar hairiness has appeared on the trunk, legs, arms and forehead. He looks about and has an intelligent expression but does not respond in any way when spoken to. He still has to be tube fed but can swallow if one is dexterous enough to insert food when his mouth is open. His arms, and especially his legs, are still somewhat spastic and the right leg shows contracture, though it is now possible nearly to straighten it without his evidencing much discomfort. Being naturally optimistic, we are expecting his ultimate recovery.

CASE 6.—The patient (48) 20 years old, became ill while he was in a hospital being treated for flatfoot. The onset, which was sudden, came on February 13, with severe pain in the chest, side, and shoulder, temperature of 102 F., pulse from 80 to 100. February 14 the condition was about the same except that he complained of pain in his eyes and during the night he became irrational. On the 15th and 16th the condition became worse, with severe headache and active delirium. His jaw was so rigid that his throat could not be examined and the arms and legs became spastic. There was slight rigidity of the neck and the Kernig sign was present, which may have been due to the general spasticity. The patellar reflexes were present, the pupils reacted rather sluggishly to light, there were a few large, moist râles in the right base posteriorly and no bronchial breathing. The pulse was rapid and of poor quality at times. His temperature went as high as 104 F. A diagnosis of cerebrospinal meningitis was made and an unsuccessful attempt was made to perform a lumbar puncture. The patient was sent to the Willard Parker Hospital on the evening of February 16. Twenty c.c. of clear fluid were withdrawn showing a moderate increase in cells and mononuclears, albumin and globulin greatly increased, and a normal Fehling's reduction. There were no organisms by smear or culture and a negative Wassermann reaction. The second puncture, February 19, showed practically the same picture except that there was a greater increase in cells. The blood count was normal, 10,000 leukocytes, 65 per cent. polymorphonuclears. His temperature ranged from 103 to 105 F. His pulse was between 120 and 130, but was not irregular until slightly before his death, which occurred February 20. He lay on his back with his eyes tightly closed and his whole body markedly spastic. His neck was rigid. It was impossible to obtain the knee jerks, but the plantar reflexes were increased. At times there was a tremor, especially of the legs. He was generally comatose but at intervals there was muttering delirium, and he frequently perspired freely. He was able to swallow until near death though he often refused unless urged to do so by one of the nurses whose directions he usually followed. His death was described as being particularly

painful with evidences of respiratory difficulty, probably of central origin, since it did not develop until shortly before he died. There was no evidence of paralysis at any time. A necropsy was performed which showed marked congestion of all the organs. The report on the brain and upper part of the cord will be given under pathology.

#### CLINICAL PICTURE

The characteristic clinical picture of lethargic encephalitis is generally as follows: a gradual onset, often following influenza, a low irregular fever, headache, marked lethargy and asthenia, with or without cranial nerve palsies.

#### TREATMENT

While there is no specific treatment, a lumbar puncture has been followed in many cases by temporary improvement. The spinal fluid is usually under increased pressure and it seems to me desirable to relieve it. Of course, every effort should be made to keep the patient comfortable and general eliminative and supportive measures should be carried out. Symptomatic treatment should be instituted as the indications arise.

#### LABORATORY FINDINGS

The blood picture is not characteristic. It is usually normal or shows a slight leukocytosis, perhaps up to 15,000. Blood cultures are sterile. The urine is usually negative (Table 2). The spinal fluid shows practically the same picture as in poliomyelitis. The cells are usually slightly or moderately increased, seldom greatly, perhaps up to 150 to 200 in some cases. While cell counts may run higher in poliomyelitis, the great majority do not. As in poliomyelitis, there is usually an excess of mononuclears, but an excess of polymorphonuclears may occur. The albumin and globulin are greatly increased, the reduction in Fehling's is normal. The increase in cells and protein content is not always in the same ratio. No organisms are shown by smear or culture. The gold chlorid curve depends on the amount of albumin and globulin present and duplicate curves may be selected from those in poliomyelitis fluids. In some instances, most often in convalescent or mild cases, the findings may depart little from the normal. This is true also in poliomyelitis. Most reports of encephalitis show that the cell count (which, unfortunately, is often the only information given) falls off very quickly. In two of our cases of long duration, the character of the fluid did not change materially over a period of several weeks, but the condition of the patients also showed little change. This comparison with poliomyelitis is made, not because I believe the two diseases are at all identical, but to emphasize the fact that in each instance the spinal fluid is not specific, but shows the

reaction of the meninges to an inflammation of the brain substance. A somewhat similar condition exists in the various syphilitic involvements of the central nervous system, but in these conditions the gold chlorid curve and the Wassermann test are helpful in making the diagnosis.



Fig. 1.—Meninges of the anterior fissure of the cord, showing fibrosis and round-cell infiltration, mostly in the vessel walls. Diffuse round-celled infiltrations of the arcuate nucleus of the pyramids is shown in either side.

The most difficult and the most needed diagnosis is that made from the fluid of tuberculous meningitis. While generally the number of cells and the increase in albumin and globulin is greater in the latter disease, it is by no means always so, and it is sometimes necessary to examine more than one fluid before one can be certain of the diagnosis, as it is often difficult to find the tuberculosis bacillus in early tuberculous meningitis and the reduction of Fehling's may be normal at that time. The following table shows the findings in the spinal fluids in our cases of lethargic encephalitis.

TABLE 2.—LABORATORY FINDINGS IN CASES OF LETHARGIC ENCEPHALITIS

Case No.	Amount in C.e.	Cytology	Protein	Fehling's Reduction	Animal In- oculation	Wassermann Reaction	Onset to Puncture	Gold Chlorid
14	30	Greatly increased..... Mononuclears 80%	+++	+++	.....	.....	?	
388	25	Greatly increased..... Mononuclears 90%	++++	.....	—	.....	3 days	
368	15 sl.	Greatly increased..... Mononuclears 90%	++	+++	.....	.....	3 days	
32	cloudy 60	Greatly increased..... Mononuclears 80%	++ 1	+++	—	—	14 days	1234321000
	35	Greatly increased..... Mononuclears 80%	+++	+++	.....	.....	15 days	
	30	Slight to moderate in- crease, Monos. 90%	+++	++	.....	.....	16 days	
	30	Greatly increased..... Mononuclears 80%	++++	++	.....	.....	18 days	
5								
9	30	No increase.....	+ 1	+++	.....	.....	5 days	
371	10	Greatly increased..... Mononuclears 80%	++	+++	.....	.....	18 days	
94								
49	12	Slight increase.....	±	+++	.....	.....	4 days	
54	5	Slight increase.....	+ 1	+++	.....	.....	5 days	
16	30	No increase.....	+	+++	.....	.....	?	
87	20	Very great increase..... Mononuclears 90%	+	+++	—	.....	4 days	
351	20	No increase.....	++ 1	+++	.....	.....	4 days	
137	10	No increase.....	+ 1	+++	.....	.....	3 days	
391	25	Slight to moderate..... Mononuclears 90%	+ 1	+++	—	—	7 days	
395	15	Slight increase.....	+	+++	.....	.....	4 days	
19	35	Moderate increase..... Mononuclears 90%	++	+++	.....	—	4 days	
41	20	Greatly increased..... Mononuclears 95%	++ 1	+++	.....	.....	3 days	
80								
357	35	No increase.....	+	+++	.....	.....	14 days	
46	15	Moderate increase..... Polys. 60%	++	+++	.....	—	3 days	
	25	Bloody fluid.....	++	+++	.....	.....	4 days	
	30	Bloody fluid.....	++ 1	+++	.....	.....	5 days	
	20	Moderate increase..... Mononuclears 80%	+++	+++	.....	.....	6 days	
	20	Moderate increase..... Mononuclears 80%	++	+++	.....	.....	8 days	
	15	Moderate increase..... Mononuclears 95%	++	+++	.....	.....	32 days	
	20	Slight to moderate.....	+++	+++	.....	.....	50 days	
35	10	Moderate increase.....	+++	+++	.....	—	2 days	
100	20	Very great increase..... Mononuclears 90%	++++	++	—	—	2 days	
	30	Great increase.....	+++++	+	.....	.....	4 days	
	15	Slight increase.....	+	+++	.....	—	4 days	
	25	Slight increase.....	+	+++	.....	.....	6 days	
331	30	Slight increase.....	+	+++	.....	.....	21 days	
382	20	No increase.....	+	+++	.....	—	1 day	
Y								
24	25	Greatly increased..... Mononuclears 80%	++ 1	+++	.....	.....	14 days	
48	20	Moderate increase..... Mononuclears 60%	+++	+++	.....	—	?	
	20	Greatly increased..... Mononuclears 80%	+++	+++	.....	.....		
353	5	Slight increase.....	++	+++	.....	.....	3 days	
393	25	Moderate increase..... Mononuclears 85%	++ 1	++ 1	—	—	10 days	
66	30	Moderate to great incr.	++ 1	+++	.....	—	15 days	
103								
27	30	Moderate increase..... Mononuclears 80%	+ 1	+++	.....	—	2 days	1232100000
59	25	Moderate to great incr.	++ 1	+++	.....	—	14 days	
72	25	No increase.....	+ 1	+++	.....	.....	3 days	
392	20	Greatly increased..... Mononuclears 90%	++	+++	—	—	10 days	
	25	Greatly increased..... Mononuclears 80%	++	++ 1	.....	.....	18 days	
	35	Moderate increase..... Mononuclears 80%	++	+++	.....	.....	20 days	
	20	Moderate to great incr. Mononuclears 90%	++	+++	.....	.....	44 days	
68								
X	30	Slight increase.....	+++	+++	.....	—	7 days	00012455560
52	30	Moderate increase.....	+	+++	.....	—	17 days	

The inoculation of monkeys with the emulsified brain and cord of fatal cases has given far from uniform and conclusive results. The English reported failures in their attempts, though the same workers had been almost uniformly successful in dealing with material from cases of poliomyelitis. Von Weisner reported an instance in which the infected monkey died in forty-six hours, his brain on necropsy showing gram-positive cocci. The short period of incubation and the



Fig. 2.—Meninges of the cortex showing marked injection of the vessels and hemorrhage invading the cortex for a short distance. The cortical tissue otherwise is negative.

finding of the organisms make it much more probable that the monkey died from bacterial infection than from encephalitis. Flexner reported inconclusive results; Strauss, Hirshfeld and Loewe have published a preliminary report that indicates that the disease may be reproduced in monkeys by inoculation. I regret that up to the present time the lack of monkeys has made it impossible for us to do any work along this line at the research laboratory.

## PATHOLOGY

Lethargic encephalitis belongs to the class of inflammatory diseases, in which also are included poliomyelitis, syphilitic lesions of the central nervous system and trypanosomiasis. While these different diseases have, broadly speaking, certain characteristics, the cases in a given class differ so widely that it is difficult, if not impossible, to accurately diagnose, by a study of the pathology alone, the less typical cases.

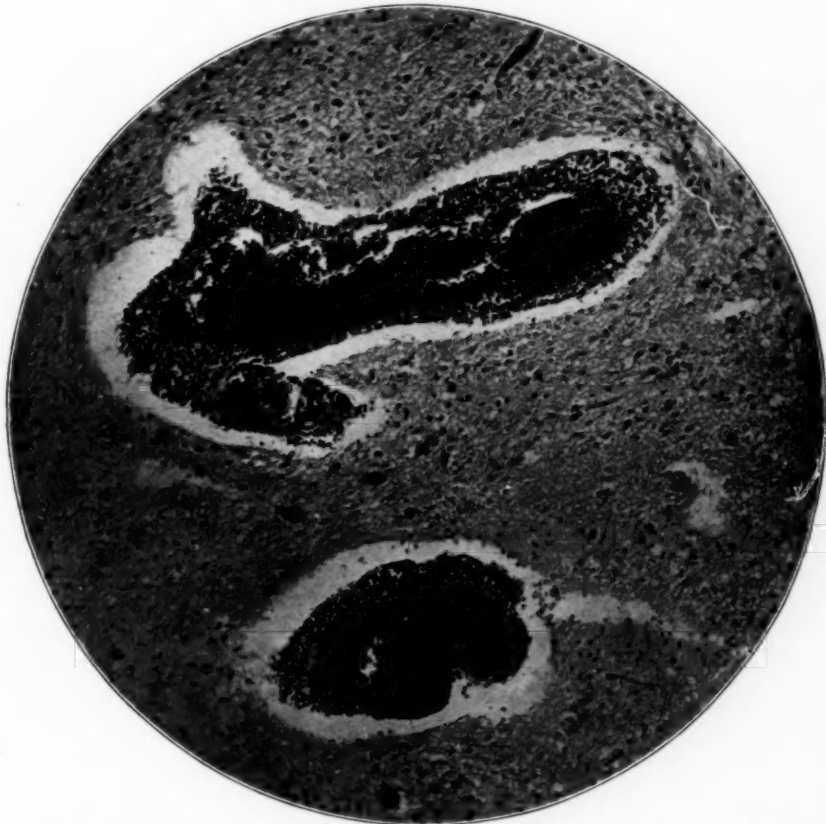


Fig. 3.—Cord, just below the olivary body, showing dense perivascular, round-celled infiltration and edema.

The meninges are usually described as showing only slight changes—an increase in the cellular elements particularly in the neighborhood of the blood vessels of the pia-arachnoid.

The cerebral cortex is generally normal except for congestion of the vessel of the leptomeninges.

In the brain substance, the changes are most marked in the basal nuclei of the brain, the upper part of the pons and peduncles, the gray matter of the floor of the fourth ventricle, and the aqueduct of

Sylvius. The changes in the medulla and cord are often reported as less pronounced, though observers have noted the same changes occurring in the upper section of the cord. This was certainly observed in the case of 48, which came to necropsy. To the localization in the mesencephalon, particularly in the vicinity of the nucleus of the third nerve, McNalty attributes the stupor, since a lesion in this locality cuts off the afferent stimuli. The relation of the paths of the rubro-



Fig. 4.—Cord, a higher level, showing dense perivascular and also diffuse round-celled infiltration.

spinal and pyramidal tracts to the region of the nucleus of the third nerve also explains the tremor and the frequent presence of the Babinski sign. It must be borne in mind that a virus affecting the nervous tissue, although it may have a predilection for a certain part of the central nervous system, may attack any part.

The lesions are generally described as consisting of four kinds:

1. Infiltration of the walls of the small vessels with lymphocytes and plasma cells.

2. Foci of interstitial and parenchymatous infiltration with round cells. In this reaction neuroglia cells may take part.

3. Lesions of the nerve cells—usually not so extensive as in poliomyelitis, and with less neuronophagia. These lesions of the cells usually occur when the inflammatory process takes place in the gray matter, but they may develop in the absence of any inflammatory reaction. Such is the case with regard to the cells of Purkinje in the cerebellum where inflammatory changes are almost entirely absent.



Fig. 5.—Medulla oblongata showing numerous petechial hemorrhages, diffuse round-celled infiltration and degeneration of ganglion cells.

4. Foci of perivascular hemorrhage. The vessel walls are usually not necrosed.

In connection with the statement that lesions of the cells may occur in regions where there is no evidence of inflammatory reaction, it is interesting to recall that Abramson, in a very excellent study of the pathology of poliomyelitis made at the research laboratory during the epidemic of 1916, brought out the same fact in regard to the lesions of poliomyelitis.

Perhaps an idea of the pathologic picture may best be obtained by a description of a case, No. 48. The brain was studied at necropsy by Dr. Alexander Fraser of Bellevue Medical School, to whom I am indebted for the following report.

#### MACROSCOPIC

The pia-arachnoid of the whole brain, including the medulla oblongata and a small part of the spinal cord accompanying it, shows



Fig. 6.—Another section from the medulla showing larger hemorrhages.

marked congestion of the vessels and numerous small hemorrhages. The portion covering the medulla, pons and peduncles is considerably thickened and brownish-gray in color. No free exudate is present. The brain tissue is firm in consistency. On section, the cerebral hemispheres show considerable distention of the vessels with blood and an occasional small splotchy hemorrhage especially in the outer cortex.

The ventricles seem large, but contain little fluid which is of a reddish tinge. The ependyma, especially over the thalamus and floor

of the fourth ventricle is lustreless, dull grayish-white in color and "mushy." In one place in the fourth ventricle it projects into the cavity in the form of a polypoid mass. The choroid plexus of the lateral ventricles is markedly congested and in places cystic. Section of the cerebellum shows distended vessels but apparently no hemorrhages. Section of the medulla, pons, crura and basal ganglions shows very marked distention of the vessels with numerous small and a few fairly large, irregularly outlined extravasation of blood.



Fig. 7.—Section of the cord showing central canal. Note disintegration of the epithelium and exudate in the lumen.

The color of the tissue in these regions is a fine and irregular mottling of dull gray and white. These features are especially marked just beneath the floor of the fourth ventricle.

#### MICROSCOPIC

The pia of the cerebrum and cerebellum shows distention of the vessels with blood, round cell infiltration especially marked around and

in the vessel walls, thrombosis and hemorrhages. Only very rarely does the perivascular infiltration follow the vessels into the brain substance, and then only for a short distance. Occasionally, a hemorrhage from one of these vessels is seen in the outer part of the cortex. In such areas the ganglion cells show various degrees of degeneration.

In the medulla, pons and basal ganglions, the pia shows the same pathologic features, but in a much more marked degree. In these

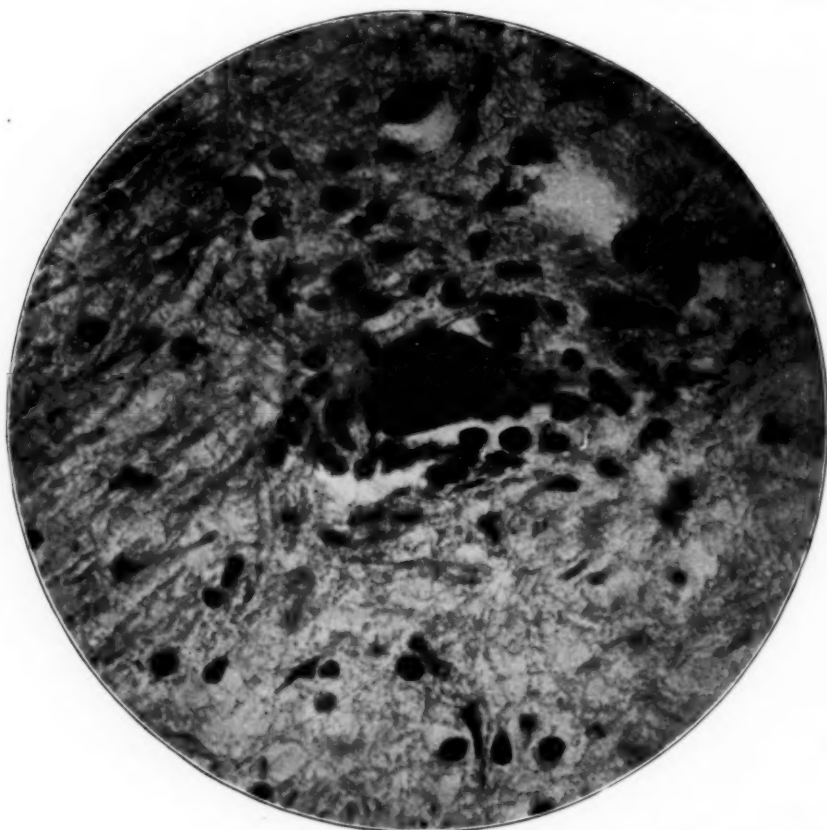


Fig. 8.—Same section as Fig. 7. Ganglion cell undergoing disintegration surrounded by phagocytes.

regions, too, the perivascular infiltration follows the vessels deeply into the brain tissue, and focal and diffuse areas of round cell infiltration are scattered throughout the tissue apart from the vessels. Frequent small and occasional large extravasations of blood are seen anywhere, but especially in the gray matter.

The cellular infiltrations, too, are practically always in the gray matter. The cells of the infiltrate are mostly lymphocytes with a few plasma cells and an occasional large mononuclear.

The blood vessels, though densely infiltrated, show no endarteritis.

In the affected areas, ganglion cells are seen in all stages of disintegration, some having been completely destroyed and replaced by groups of phagocytes. Sections stained by the Levaditi method were negative.

#### CONCLUSIONS

1. The histologic picture of the condition is that described by English authors for lethargic encephalitis, also that of trypanosomiasis.

2. The picture is very much like poliomyelitis, but such extensive infiltration of meninges and larger vessel walls would, at least, be very unusual.

3. The picture is also very much like syphilis. In syphilis, however, the infiltration sticks to the vessels. Syphilis shows an endarteritis, and usually gummata in vessel walls.

#### POSSIBLE RELATION TO POLIOMYELITIS AND INFLUENZA

Three theories have been advanced to explain the occurrence of lethargic encephalitis. When it first appeared in England, it was suggested that it was caused by food — botulism or some poison derived from substitutes or solanin accumulating in sprouts of potatoes or other vegetables. This theory has been definitely disproved and discarded. According to a second theory, it is a form of poliomyelitis; and, according to a third, it is connected with the epidemic of influenza.

The theory that it is a form of poliomyelitis has not been definitely proved or disproved. Epidemic poliomyelitis usually occurs in hot weather, the majority of the victims are children, and the lower motor neuron type of paralysis constitutes the great majority of the cases with paralysis. The onset is usually sudden and the greatest number of deaths occur in the first week. Lethargic encephalitis has occurred in its present appearance during the cool weather, the majority of cases having been adults (my own list of cases shows a large number of children, but this is undoubtedly because I am so often called to see the milder type of case where tuberculous meningitis is suspected). Very few cases of the lower motor neuron type of poliomyelitis are occurring, and among the cases diagnosed as lethargic encephalitis there are evidences of involvement of the higher centers in the way of cranial nerve palsies and prolonged lethargy which are rare even in epidemics of poliomyelitis. Certainly among the hundreds of cases of the latter disease that came under my observation during the epidemic of 1916 there were no cases at all approaching the characteristic pic-

ture of lethargic encephalitis, and only a few of the encephalitic type of poliomyelitis, with which some of these milder cases might easily be confused. Moreover, in lethargic encephalitis the onset is usually slow, and death occurs oftenest in the third week. The similarity of the spinal fluid findings is of little significance, since in neither case are they specific. The same may be said in regard to the pathology, though here there are, as a rule, more points of difference. As regards animal inoculation, it is certainly much more difficult to reproduce the disease in monkeys than is the case in poliomyelitis. For all these reasons it seems to me probable that lethargic encephalitis is not a form of poliomyelitis though the causative agents in the two diseases may perhaps be closely allied.

In regard to the possible relation between influenza and lethargic encephalitis, the evidence is as yet entirely circumstantial. In the first place, attention may be called to the fact brought out by historical study that on several occasions epidemics of a disease resembling lethargic encephalitis and influenza have occurred together. The impression is gained from these studies that encephalitis has not appeared in anything like an epidemic form except with influenza. It is certain that in their last appearance, 1889-1890, they occurred simultaneously, and it would seem that enough time has elapsed since for either to appear by itself if there were no direct connections between them. Then again, in a large proportion of cases, occurring in this country at least, the onset has been preceded by an attack clinically influenza. Moreover, that influenza has a marked effect on the central nervous system is shown in two ways: First, in nearly every instance, the convalescence from influenza is characterized by a profound mental depression and nervous exhaustion out of all proportion to the severity of the disease; secondly, as indicated by the reports of Jelliffe, Menninger, Burr and others, influenza is far more likely than any other acute infection to be followed by disturbances of the nervous system either psychic or organic. Therefore, it seems to me probable that there is a definite connection between influenza and lethargic encephalitis. Just what the relation is, I am not prepared to state. In view of the fact that we do not know the cause of either disease, one cannot say that the two diseases have the same origin. The causative agents may be identical or closely allied or the virus causing influenza may make the individual more susceptible to the causative agent of encephalitis or it may enhance its virulence. It is easy to speculate on the unknown.

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## DISCUSSION

DR. JOHN F. HOGAN, Baltimore: In Baltimore we wished to ascertain the number of cases of encephalitis lethargica, as only a few were diagnosed. I saw all of the cases reported to the health department and there was one constant symptom or rather complication. I think it should be classed as a complication rather than a symptom, because it comes on later in the progress of the disease. This constant complication of diplopia seemed to set in after-

ward, within three to five weeks after the onset. In all the cases that I saw, the complication was present or developed later. The first patient I saw was a big husky man, 20 years of age. I did not know what his trouble was. He had been sent to a camp in that vicinity because he was a member of the Dental Reserve Corps. In February or March I heard that this man had recovered. He had had a slight facial paralysis which afterward disappeared, and as I questioned him and read the article to him, which appeared about that time in the report of the Surgeon-General of the U. S. Public Health Service, and I described diplopia he said he had it. He spoke of attending a lecture at the camp and while at the lecture he thought he saw two lecturers on the platform and, in fact, questioned a soldier who was next to him and asked him if there were two persons on the platform. I wonder if others had the same experience.

## PSYCHOSES ASSOCIATED WITH INFLUENZA

### II. SPECIFIC DATA. AN EXPOSITORY ANALYSIS \*

KARL A. MENNINGER, M.S., M.D.

Assistant in Neuropathology, Harvard Medical School; Assistant in Neurology,  
Tufts Medical School

TOPEKA, KANSAS

Psychic disturbances associated with influenza are first mentioned in the literature in connection with an epidemic of 1385 in Germany.<sup>1</sup> The "deliria" of the epidemic of 1387 are mentioned in accounts<sup>2</sup> by Valescus de Taranta and Gassar. The "vexatious deliria" of the epidemic of 1510 are mentioned by Mezèray.<sup>3</sup> In "Annals of Influenza,"<sup>4</sup> published in 1852, Riverius<sup>5</sup> is quoted regarding the epidemic of 1580 thus: "It began with a fever and cough, then followed again a pain of the head and loins, then the fever intermitted a few days and returned with fresh vigor. Some had no rest, but the heat increasing they died; as some did of a phrenzy (!) and others of a consumption." Henisch the First spoke of the extreme prostration, "somnolent states, lipothymias, and other disquieting incidents" of this same epidemic. Quoted by Espagnol,<sup>6</sup> Ozanam<sup>7</sup> recounts the occurrence of "such grave symptoms as convulsive movements and somnolence" after the disease in the epidemic of 1691. Schweig<sup>8</sup> described an epidemic in 1737 with psychic disorders similar to those he found mentioned in connection "Maladie du Sommeil," because of the somnolent and stuporous states following it (1718).

\* Contribution I was published in J. A. M. A. 72:235 (Jan. 25) 1919.

\* A contribution from the Psychopathic Department, Boston State Hospital series of 1919.

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8. Schweig: Die Influenza, Berlin, 1836.

9. Camerarius: Quoted by Espagnol, without reference.

But despite these and other mentions of the concomitant deliria and numerous neurological manifestations, it remained for an American to signalize the association of "symptoms of madness" and "loss of reason" with influenza. This was Benjamin Rush of Philadelphia, in "An Account of the Influenza as it Appeared in Philadelphia, 1789, 1790 and 1791."<sup>10</sup> Thereafter there were sporadic reports, for example, by Bonnet in 1837 "of an individual in whom the grippe provoked a furious mania"; by Pétrequin, who spoke of "patients tormented by sad ideas," and of "four or five suicides . . . in the hospitals of Paris" in the epidemic of 1837; by Chrichton-Brown in 1874, of "acute dementia following the grippe."<sup>11, 12, 13</sup>

The epidemic of 1890 was evidently followed by many cases of mental disease, and the increased interest in psychiatry at this period brought forth a shower of literary progeny. Innumerable articles and reports appeared then and thereafter relative to the association of influenza with mental and nervous disease. Of the hundreds of names which are available in any complete bibliography, a few stand out as of preeminent importance and are still frequently quoted. Thus Kraepelin<sup>14</sup> and Bonhoeffer<sup>15</sup> are known because of their later prestige; Leichtenstern<sup>16</sup> for his comprehensive study of influenza as a whole; Kirn<sup>17</sup> because of his numerous able contributions; Ladame,<sup>18</sup> Bidon,<sup>19</sup> and others of the French school because of the different viewpoint represented by them; and Leledy<sup>20</sup> and Bossers<sup>21</sup> for thorough historical and bibliographical records of the association of influenza and

10. Rush: *An Account of Influenza as It Appeared in Philadelphia, 1789, 1790 and 1791. Inquiries and Observations*, Philadelphia, 1805, p. 353.

11. Bonnet: *J. de méd. de Bordeaux*, 2nd series, Part 5, p. 175, 1837.

12. Pétrequin: *Gaz. méd. de Par.* **51**:801 (Dec. 23) 1837.

13. Chrichton-Brown, J.: *West Riding Lunatic Asylum Medical Reports* **55**:269, 1874.

14. Kraepelin, E.: *Ueber den Einfluss acuter Krankheiten auf die Entstehung von Geisteskrankheiten*, *Arch. f. Psychiat.* **11**: 1881, and **12**: 1882. Also, *Ueber Psychosen nach Influenza in Dorpat*, *Deutsch. med. Wchnschr.* **11**: (March 13) 1890.

15. Bonhoeffer: In Aschaffenburg's *Handbuch* (Leipzig and Vienna), 1912.

16. Leichtenstern, O.: In Nothnagel's *Encyclopedia*. "Influenza." English translation by Stengel, 1905, Philadelphia, W. B. Saunders Co.

17. Kirn: *Ueber Influenza Psychosen*, *Munchen. med. Wchnschr.* **37**:299-301, 1890. *Die nervoesen und psychischen Störungen der Influenza*, *Samml. klin. Vortr.*, n. F. **23**: Leipzig, 1891; *Die Psychosen der Influenza*, *Allg. Ztschr. f. Psychiat.* **48**:1-15, 1891-1892.

18. Ladame: *Annal. méd.-psychol.* **13**:20, 1896.

19. Bidon: *Revue de Médecine*, 1890.

20. Leledy, A.: *Lagrippe et alienation mentale*, 4, Paris, 1891.

21. Bossers, A. J.: *Die Geschichte der Influenza und ihre nervoesen und psychischen Nachkrankheiten*, Leiden, 1894.

psychosis. (The historical summary of the first paragraph of this paper is based in large part on these accounts.) In addition there were a great number of current literature accounts — about 220 authors are listed in the Surgeon-General's Index Catalog, 1902 volume. Books on the subject were issued in German by Ahrens, Borchardt, Bossers, Brachmann, Eberlings, Hirschfeld, Jutroinski, Klemm, Lochner, Mispelbaum, Mucha, Wescher and Weynerowski; in French by Auguin, Biet, Brionne, De Groote, Diemer, Espagnol, Lehmann, Le Joubieux, Leledy, Lestra, Ulliel, Trastour, and Virey; in Danish by Fehr, and in Italian by Rossi. I know of no books in the English language.

All of these accounts have a certain intrinsic interest, but many of them are of dubious value to the present day psychiatrist because of the ambiguity of the antiquated terminology. The insistence on interpretation instead of pure description also militates against the usefulness of many of the case histories, and the original meanings of many of the terms used have now become obsolete. This may be illustrated by the extreme elasticity and vagueness, even today, of the protean term "delirium."

#### THEORY OF THE SPECIFICITY OF MENTAL SYNDROMES

Since 1890 the reorganization of nosologic classification, the animadversions of freudianism and psycho-analysis, and the freedom from pandemics have combined to keep literature on the mental side of influenza almost negligible in amount. There have been a few contributions, such as that of Gosline,<sup>22</sup> Jelliffe<sup>23</sup> and others, but none has made any claim to exhaustiveness. There has been, on the other hand, rather a tendency to generalization and to the assigning of categorical principles even before the complete assembling of pertinent details. Thus arose the question of the specificity of mental syndromes with acute infectious diseases, ardently sponsored by Kraepelin and vigorously combated by Bonhoeffer and the French school. Kraepelin's prestige was sufficient to project, even in the face of majority opposition, the thesis that each acute infectious disease could, in the course of further study, be recognized from the mental symptoms alone. This, in short, is equivalent to declaring for the autonomous entity of a variolar psychosis, a typhoidal psychosis, an influenzal psychosis, etc. Except in the case of pellagra (an exceedingly dubious exception), this possibility is not yet recognized, nor is there any general tendency to ascribe the fact that it is not to our failure to read all the signs, as Kraepelin maintained.

22. Gosline: Newer Conceptions of Dementia Praecox, *J. Lab. & Clin. Med.* 2:691 (July) 1917.

23. Jelliffe, S. E.: Influenza and the Nervous System, *Phil. Med. J.* 1041 (Dec. 27) 1902; Psychoses with Influenza, *N. Y. Med. J.* 108:725 (Oct. 26) 1918.

It cannot be gainsaid, however, that influenza apparently produces an exceptionally potent neurotoxin. After the last great epidemic, in 1890, Leichtenstern<sup>10</sup> in his celebrated monograph wrote "This epidemic has taught us something quite new in neurology, namely, an acute infectious disease which compared with all others is characterized by its striking neurotoxic effects. The sentence . . . 'The influenza toxins are intense nervous poisons' has found full justification in the history of earlier as well as in the latest epidemics." Most writers agree with Leichtenstern that influenza is preeminent as an inciter of nervous system sequelae, although Bonhoeffer<sup>15</sup> and a few others believe in the supremacy of typhoid. But even to the present time there is no proof that these sequelae are qualitatively specific.

#### THE QUESTION OF PREDISPOSITION

A corollary question arose which is in a sense more important. This is the question of nervous predisposition as a soil for the psychopathies and neuropathies which arise after influenza. It was Leichtenstern's opinion that "influenza produces a specific nervous toxin which, besides producing conspicuous nervous prostration, in some cases, by its poisonous action on the cortex of the brain, calls forth these psychoses." He points in proof to the two facts, that (1) "the cases observed by us were conspicuous by the fact that hereditary or other neuropathic influences were not found in them, and (2) the obvious frequency with which the young, even infants, were affected by these psychoses." Opposing this view were many writers: Ladame, Mehr, Mispelbaum, Ayer, Justrosinski, Kraepelin, et al. Kraepelin took herein a rather self-contradictory view, for despite his generic theory of specificity, he declared in regard to this particular disease that "it would seem that influenza alone would not be able to produce a disturbance of the mental function in a normally constituted person." Schuele echoes this: "Influenza only plays the part of an etiologic accompaniment in the production of mental disease." And Ladame, "Influenza by itself is never sufficient to produce insanity." Leichtenstern marshals Althaus, Bossers, Mucha and Ulliel as authorities admitting of the possibility of psychoses arising from influenza alone, without "nervous predisposition," and quotes Kirn as having found evidences of predisposition in only 23 per cent. of the *cum-febrile* psychoses, but in 92 per cent. of the postfebrile psychoses. He attacks the evidences for predisposition in the latter, however, and, to the writer, seems justified.

At present these questions remain much as at the time of this discussion twenty years ago. The recent pandemic of influenza stands in a fair way to settle them. Of the important acute, infectious dis-

eases occurring in epidemics, typhoid and variola are so well controlled that any large mass of material from which to extract psychiatric data is not to be expected. Plague has not of recent years been epidemic, and the present advance of preventive medicine is not likely to permit it to become widespread. Influenza alone remains, and no comment is necessary to remind us of its ubiquity.

#### THE BOSTON SERIES

About 120 cases of mental disease with a history of recent influenza were admitted to the Boston Psychopathic Hospital during the last three and one-half months of 1918. (About sixty additional cases were seen early in 1919, and will be presented in future communications.) A statistical analysis of eighty of this group was recently presented<sup>24</sup> as the first of a series of papers dealing with the subject from this hospital. I pointed out then that on the basis of statistical indications, the prominent points brought out were the frequency of schizophrenia (dementia praecox) as a postinfluenzal psychosis, the difficulty in diagnosis between delirium and schizophrenia in many cases, the wide variety of psychotic syndromes observed and the conspicuous absence of stigmata of mental predisposition in the majority of the cases.

The answers to the two questions of specificity and of predisposition are thus strongly suggested. But these answers may not be learned from data purely statistical. For one thing, they fail to consider certain essential mathematical fallacies. Thus, 10 per cent. of cases observed in this hospital may have been deliria, but this in no accurate way represents the relative frequency of delirium in influenza or its frequency as a form of mental syndrome accompanying influenza.

Consequently, further study of the data accumulated is expedient, and is herein set forth. Of various modes of attack on the problem of "What forms of mental disease result from influenza?" an expository analysis by case exemplification lends itself with particular facility. The 120 cases of psychoses in the production of which influenza was an apparent factor, embrace types of all the recognized psychiatric groups, and the data of typical cases constitute the most accessible and forceful negation at hand of the theories of specificity and essential predisposition. The variety of psychiatric manifestations and the varying rôles of influenza, primary, secondary and collateral, appear from the following representative cases.

24. Menninger, Karl A.: Psychoses Associated with Influenza. I. A Statistical Analysis, *J. A. M. A.* **72**:235 (Jan 25) 1919.

In the presentation of these I have chosen to follow the classification of Southard.<sup>25</sup> His regrouping of the major forms of mental disease into eleven large categories has the great advantage of convenience. This virtue appears in a comparison of the facility of illustrating the main types of mental disease encountered in the present series. For while it would be quite feasible to illustrate at least twenty of the twenty-two of the psychoses of the American Medico-Psychological Association list (pellagra psychosis, for example, omitted) with influenzal psychoses, it would in no way amplify, except in bulk, the contribution of an illustration of each of the eleven groups of Southard. Without further comment, these appear.

#### ILLUSTRATIVE CASES

##### GROUP 1.—*Syphilopsychoses. Neurosyphilis*

The precipitation of the psychoses of neurosyphilis by acute infections is well known because of the spectacular results rather than because of its frequency. There is a surprising paucity of references to it in the literature, however. The following is one of a series of this type.<sup>26</sup>

##### CASE A.—*Latent Neurosyphilis + Influenza = Active Paresis.*

*Family History.*—The patient was an unmarried man, aged 46, a watchman by occupation, born in the United States. The family history was entirely negative, including an account of the grandparents, uncles, aunts and siblings, except that there were three siblings who died at birth.

*Past History.*—He had evidently been a healthy, normal child, and graduated from grammar school at 14, entering economic life as a shoe-store clerk. Subsequently he worked in the city fire department. At the age of 32 his thigh and hip were fractured in an accident, and he was given a position as customs guard. He worked steadily until the day he went to bed with influenza. There is no court record. He used no alcohol and no tobacco. Except for the fractured hip his medical history is negative. In disposition he is described as being happy and sociable, with a fondness for music and the theater. The only previous symptom of any kind which was elicited was that he had complained of being tired during the past four summers.

*Present Illness.*—September 20, he contracted influenza, which evidently attained a rather severe degree. At one time the temperature was reported as 104 F.; but returned to normal on the fourth day. He arose from his bed and was up and about the house, planning to return to work, but seemed to contract "a cold" again and returned to bed two weeks after arising. He was in bed part of the day, but was up the next day, feeling quite well again. That night he became "delirious." He is described as having been irritable and having refused to take the medicine from the doctor, declaring that it

25. Southard, E. E.: The Genera in Certain Great Groups or Orders of Mental Disease, *Arch. Neurol. & Psychiat.* **1**:95 (Jan.) 1919.

26. Menninger, K. A.: Influenza and Neurosyphilis, *Arch. Int. Med.* **24**:98 (July 15) 1919.

was the wrong kind. He hung his head over one side of the bed and let it so remain. He refused to do anything he was told, and announced that the Blessed Virgin Mary was going to tell him what to do. About this time occurred, according to his wife, one—possibly two—brief seizures. He was brought to this hospital, the admission not provoking any severe psychic reaction.

*Mental Examination.*—(Summarized.) His general attitude was that of accessibility and cooperation, although he was at times self-absorbed and agitated, or depressed. His *sensorium* seemed to be entirely clear. His *orientation* was precise, except that he had no memory of how long he had been in the hospital. His *memory* was grossly defective. Not only were many facts of his past history forgotten, but details of all kinds were lost, and his memory for recent events was extremely poor. He recognized short periods of total amnesia occurring during the previous two weeks. His ideation showed fleeting persecutory delusions without systematization, and certain delusions of other types—ideas that his mother was dying, that the Virgin Mary would guard him, that misfortune was about to overtake his family. No hallucinations were elicited. His *emotional tone* showed, as stated above, instability and, at times, a tendency toward mild, agitated depression; he frequently wept. The *thought processes* were badly disrupted, but there was no blocking, retardation or acceleration. His *motor activity* was slightly decreased.

*Physical Examination.*—This showed the patient to be a poorly nourished, poorly developed man, aged 46, 5 feet, 7 inches tall, weighing 130 pounds. Special senses, epicritic and protopathic sensory interpretations were normal; but deep sensation somewhat impaired. There was no Babinski and no ankle clonus, but a slight swaying in Romberg's position. Knee jerks were equal and active. Pupils were contracted, fixed, and reacted neither to light nor to distance. Corneal and pharyngeal reflexes were present; extra-ocular movements, normal; abdominal and cremasteric reflexes, absent. There was tremor of facial muscles, tongue and extended hands.

*Laboratory Findings.*—The urinalysis was negative. Blood serum Wassermann test, positive. Spinal Fluid: Albumin, ++; globulin, ++; cells, 41; colloidal gold reaction, 5555555555. Wassermann test, positive.

*Diagnosis.*—General paresis. He was committed.

#### GROUP 2.—*Hypophrenoses. Hypophrenia*

No positive proof in confirmation of the production of Kraepelin's dubious "infectious idiocy" in previously sound material was obtained. This term is a glaring example of Kraepelin's inaptness at nomenclature, as the expression "infectious idiocy" certainly violates all canons of correct designation of mental disease. "Infection dementia," while open to criticism, is certainly more accurately expressive of his meaning. Waiving logomachical contentions, however, we may say that there has been no present evidence of an utter annihilation of mental processes by influenza in nonfatal cases.

Subtotal mental or intellectual loss was, however, definitely demonstrated. The following remarkable case is one of several of a similar

nature observed. Burr has mentioned having seen several such in a recent report.<sup>27</sup>

CASE B.—*Morosis + Influenza = Imbecility.*

*Family History.*—The patient was a boy aged 10, of American parentage. The family history is entirely negative. An only brother, a year younger, is quite well, and is doing fair work in school; he is in the fifth grade.

*Past History.*—He was born at full term—normal delivery, and had had no severe illnesses. His teeth appeared at 5 months; he walked at 18 months; talked at 3 years, and started kindergarten at 4.

*School Record:* (His teacher was interviewed.) He did poor work in kindergarten the first year and was kept there a second year, which is only done "with particularly backward children." He was promoted to first grade, but had to repeat the work, and then, though it was still poor, he was promoted. "His work in second grade was poor but he was promoted because considered too old to stay in the grade." The third grade work was poor, and was repeated this year . . . record, so far, poor. The patient had so far had the very lowest rank given, and was promoted only because of lack of better arrangement, and because of a desire not to keep backward children behind indefinitely.

*Disposition:* He was always quiet, rather seclusive and played with childish toys which even the younger brother has outgrown. His teacher reports that his deportment is good, and that he does not seem sensitive about his backwardness. There had never been any conduct disorder of note.

*Present Illness.*—He had influenza for four days, temperature not known. He "slept without waking" for three days, taking no food. The physician and his nurse insisted on his staying in bed a week, which he did without complaint, "perfectly normal and happy" up to this time. Three weeks after the influenza (on Christmas morning) he complained of headache and fatigue, and by his parents' statement "has been out of his head since." (The history was obtained on the 28th.) He cried constantly; wandered aimlessly about; went outdoors and became lost; occasionally sang in a feeble voice numerous popular songs; at other times screamed and cried and refused to remain in bed; said, "I'm lost. Take me home. I will be good to you." His parents were most alarmed because he failed to recognize his mother. He was brought to the outpatient department and recommended to the house.

*Mental Examination.*—For the first two days he was constantly blubbering and wailing, the more so when any attempt was made to comfort or quiet him. He selected one of the nurses finally, whose attentions he would permit, and dragged himself about the ward at her heels thereafter, crying when any one else approached him. He did not scream petulantly, but cried as if in grief and begged to be taken home to his mother. He did not express delusions or hallucinations. He was sleepless for the greater part of the first four nights, but gradually slept better at night and cried less in the daytime until, on the fifth day, he was quiet all day, clothed, accessible, and, in a faint way, cooperative. *Orientation* for place, time and person was retained from the start. His *memory* defect was most prominently shown in school learning, his knowledge of more general matters being more open to question than his memory thereof. He was tested by the tests used at the Waverley

27. Burr: Mental Complications and Sequelae of Influenza, *Med. Clin. of N. A.* 2:709 (Nov.) 1918.

School for the Feeble-minded and found incapable of doing any of the third grade tests, many of the second grade tests, and not a few of the first grade requirements. Thus, to select some of the most glaring deficiencies, he could not count by twos, he could do no subtraction at all, he misspelled "do," "go," "run" and other words little more difficult. He did spell correctly "cat," "it," "and," and "an," and did a few simple problems in addition, such as "2 + 3, 1 + 4, and even 3 + 7," but he could not get much above the latter. *Ideation* was represented by an absence of delusions and an absence of insight. He gave a very inadequate account of his recent trouble, although he did not seem to have had any amnesic periods. *Hallucinations* were not proved. *Thought processes* showed no defect of attention after the fourth day; the associations which were rather slow were not schizophrenic, incoherent or erratic, although of course quite puerile. *Emotional tone* was, of course, a point in question. He was very lonely and nostalgic at first; whether or not this accounted in any considerable measure for his conduct is questionable. During the examination he showed no emotion except when home was mentioned, which evoked a few quiet tears. On the day of discharge he was quite elated and happy because he was going home. *Motor Status*.—His conduct disorder was confined to the weeping and wailing and screaming of the first few days.

*Subsequent History*.—On the day of discharge—January 4—his lugubriousness was replaced with smiles and complacency. His memory was again tested briefly and found unimpaired, even for the recent episode; insight, essentially lacking, although he did say that his head was "not right" and that he had been "dizzy from rocking too much;" intellectual processes, definitely, but slightly improved; ideation, as before, and conduct, above reproach. But he took no interest in trying to do ward work nor gave other manifestations of normal initiative.

*Physical Examination*.—He was a rather undersized lad whose physical examination was negative in all respects save for "a suggestion of hutchinsonian teeth." These were the incisors, particularly the uppers, which had an elliptical or concave contour of the margin, not typically hutchinsonian.

*Laboratory Findings*.—Urine negative. Blood serum Wassermann test negative. Spinal fluid negative, including Wassermann test.

*Psychometric Tests*.—The patient graded regularly, variation total of 7, at a mental age of 5.7 on the Yerkes-Bridges Point Scale. On the Stanford Scale, four days later, he graded at 6 years, 2 months. In the supplementary tests his performance in the construction puzzles was poor. The memory tests were also poorly done, and he accepted ten out of ten suggestions. The patient cooperated poorly on the Point Scale and fairly well on the Stanford.

He was discharged with recommendation for instruction in a school for the feeble-minded. The diagnosis at discharge was imbecility.

### GROUP 3.—*Epileptoses. Epileptic Psychoses*

The final word on the influence of influenza on the production or augmentation of epilepsy should come from the institutions primarily devoted to the care of that disease. The first mention of the matter is probably that of Michell,<sup>28</sup> who wrote of the epidemic in Holland, 1782,

28. Michell: Antwood over de Febres catarrhales; in *Verhandelingen van het Zeeuwsch Genootschap der Wetenschappen te Vlissingen*, p. 162; Michell: *Geneeskundige Verhandeling over de . . . Febres catarrhales . . . in den Nederlanden*, Middelburg, 1875.

that it "brings about in the brain apoplexy, epilepsy, chorea and . . . convulsive movements." The direct causation of "a typical but chronic epilepsy" which, however, always recovered, has been reported by Landgraf,<sup>29</sup> Van Deventer,<sup>30</sup> Jaccoud<sup>31</sup> and Leichtenstern.<sup>16, 32</sup> Of this type we saw none whatever. Our experience has been quite contrariwise, that epileptic attacks were indeed precipitated or accelerated in known epileptics, but that in no unpredisposed case did they occur for the first time after influenza. The cases of epilepsy, curiously enough, are so generally complicated by other factors, for example, hypophrenia, parturition and appendicitis, that it is difficult to present a good type case. The following case is interesting for the unusual change after influenza from the typical convulsive seizures to the somnambulistic or twilight-state episodes, and illustrates also an increase in frequency of attacks.

CASE C.—*Occasional Epileptic Seizures + Influenza = Epileptic Twilight States and Increased Frequency.*

*Family History.*—An unmarried American sailor, discharged, aged 22, had a negative family history except that one paternal uncle is said to have had epilepsy. A brother, aged 23, is living and well.

*Past History.*—The patient was born in Maine; had a normal childhood, and received a grammar and high school education. He left the high school to go to work, but later joined the U. S. Navy and attended a radio school. He was held back one year in high school because of delinquency in algebra and English. He enlisted in the U. S. Navy in April, 1917, and was discharged in March, 1918, presumably because of his epileptic attacks. He admitted having concealed his epileptic history on enlistment, but it was discovered in the Navy and he was held for medical examination in naval hospitals for some months before discharge. Since that time he has been employed at testing steel in an arsenal.

*Medical History:* This was negative except for epilepsy. He was never seriously ill nor injured. He used no alcohol, tobacco only moderately and denied sexual perversions.

*Epileptic History.*—Attacks began at the age of 12; no particular circumstances of the first seizure could be obtained. Thereafter they occurred about every two months until a few years prior to admission. The attacks entailed the characteristic tonic convulsions, frothing, biting the tongue, etc., but were not extremely severe and, as they were preceded by a somewhat prolonged aura, he had never injured himself severely. They were always followed by a period of headache and malaise, with total amnesia for the events of the seizure. For a year or so prior to his enlistment in the Navy they had been

29. Landgraf: Gesellschaft der Charité-Aerzte in Berlin, Berl. klin. Wehnschr. 1890, Nos. 9 and 12.

30. Van Deventer: Centralbl. f. Nerven- u. Psychiat. 13:49.

31. Jaccoud: Pathologie interne, Paris, 1870, p. 780, and Nouveau dictionnaire, etc., Paris, 1873, Part 16, p. 740.

32. Leichtenstern, O.: Influenza Lectures, publ. in Deutsch. med. Wehnschr., 1890, Nos. 11, 15, 18, 22, 23, 29, 30, 42, 43.

decreasing in frequency, under the treatment of Dr. Waterman of Boston, and during the period of his Navy enlistment, almost a year, he had but two seizures. He was discharged from the Navy in March and between then and November had two or three more attacks.

*Present Illness.*—September 30 he contracted influenza and was in the Homeopathic Hospital until November 1. He had pneumonia and was not expected to live. During his acute illness he was delirious and did not remember his father's visits. He had no epileptic attacks during the month.

During November and December he had three epileptic attacks. These were of the nature of somnambulisms, however, instead of the convulsive type as previously. He was first noticed by his employer one day to be managing his duties clumsily and when addressed did not reply; so he was taken home, where he subsequently recovered without memory of the event. On another occasion he suddenly left work, taking with him a friend's letter to which he was in no way entitled, and was observed to stagger as if drunk. The third attack was the event of his coming to this hospital. He last recalls being at his work as usual; the police found him wandering the streets in an adjacent suburb of Boston, and failing in satisfactory replies, he was brought by them here. "In the admission office he has to be prodded with questions frequently in order that any answers may be elicited, and to keep him awake. Said he could not tell where he has been living in Boston the past three years. Later replies that he was a radio student at Harvard."

He was taken to the ward, fell immediately asleep, and when seen the following morning was quite clear, but could give no account of the interval between leaving work the day before and awaking in this hospital.

*Mental Examination.*—This was negative. He was a well appearing, intelligent, alert young man fully accessible and cooperative. *Memory, orientation, ideation, thought processes, emotional tone and motor status* were quite normal; there were no hallucinations and no memory of any. He was amnesic for the periods of his epileptic attacks and for the periods during which he was delirious with the influenza and pneumonia.

*Physical Examination.*—Entirely negative.

*Laboratory Findings.*—Entirely negative, including spinal fluid and serum Wassermann tests. The *psychometric test* gave a rating of 17 plus.

*Diagnosis.*—Epilepsy, twilight state. Not psychotic.

The following case, while not one of the series observed at this hospital, is rather more representative of the general tendency here observed. This case was one seen at Dr. Myerson's clinic at the Boston Dispensary, where the writer was an assistant visiting neurologist.

*CASE D.—Epilepsy + Influenza = Shower of Attacks, Then Complete (?) Cessation.*

*Family History.*—A white schoolboy, aged 14, had a negative family history as far as frank epilepsy or other mental diseases were concerned. The father, aged 45, was subject to outbursts of temper, and the mother, aged 38, to chronic headaches, but such ubiquitous symptoms should probably be disregarded. One sister is living and well. Both parents were born in Ireland.

*Past History.*—The patient was born in Boston, 1904, and except for measles, rubella and chronic tendency to constipation, had always been well. He was in the seventh grade at 13, but lost ground because of his epilepsy and was repeating the grade at 14 when seen by us.

*Epileptic History.*—In August, 1916, he had a sudden epileptiform seizure which initiated his epileptic history. This and subsequent attacks were generally preceded by auras of dizziness and "stomachache," and an aggravation of his constipation generally foreran the attacks by several days, sometimes weeks. Just before the fall he was accustomed to see the walls and floor moving.

The attacks have occurred both day and night and in all sorts of places. He has, however, never severely hurt himself, bitten his tongue, or passed urine. He lost consciousness totally and had tonic and clonic convulsions. After the attacks, which are of variable duration, he has severe headaches. Occasionally the attacks are mild.

The average interval was given as "two or three months." As he was being treated in the diet clinic for constipation and in the skin clinic for herpes zoster, the attacks were usually noted, and the following dates appear, showing greater frequency: Oct. 10, 1917; Nov. 17, 1917; Jan. 30, 1918; "Is beginning constipated again"—April 24; May 2; June 23; July 15  $\pm$ ; August 12.

*Present Illness.*—About the last week in September he contracted influenza, was very ill at the City Hospital and bedridden for a month. During his illness he had three, possibly (he himself says) four more seizures. The last was on October 21, while he was still bedridden. Thereafter he was seen in the nerve clinic frequently and had no more seizures. (Seen January 14; no further attacks up to that time.) A physical examination was entirely negative, and a Wassermann test on the blood serum was negative.

GROUP 4.—*Pharmacopsychoses.* Psychoses from drugs, alcohol, etc.

Paradigm: *Alcohol(ism) + Influenza = Delirium Tremens*

I recognize the criticism which the presentation as an influenzal process of so prosaic a phenomenon as suggested by this paradigm will provoke. Nevertheless, there is some defense. First of all, it is certainly the most frequent, and, secondly, it is by no means the best understood psychic manifestation of the combined effects of alcoholic and infectious toxemia. It is a considerable question whether the apparent is the real in the case of the suggestions of our paradigm. For all its plebeian frequency, delirium tremens seems far from being completely understood (and we have been awaiting the cry from the alcoholic business interests that the national prohibition amendment has foreclosed the possibility of further study of alcoholic psychoses in this country!). There are not a few (for example, Bonhoeffer) who do not regard the psychosis as a primary result of the alcoholic toxemia, or the direct effect of alcohol on the cortex. Nor is there adequate rationalization for both the excess and the withdrawal incidences.

In regard to the influence of influenza as a specific form of infection in the precipitation of delirium tremens, there are no conclusive data. Rosenbach<sup>33</sup> thought the epidemic of 1890 was followed by delirium

33. Rosenbach: Berl. klin. Wchnschr., 1890, p. 95.

tremens of usual frequency and gravity. But "the assumption that influenza on account of its marked neurotoxic character is relatively more frequently accompanied by delirium tremens than any other acute infectious disease, is quite unfounded. . . . The frequency of alcoholic delirium [sic] during influenza periods is noted on all sides. (Rosenbach, Nagy, Bruns and others) . . . (But this) is an absolute increase due to the enormous morbidity of influenza and especially to the frequent occurrence of influenza pneumonia." With these remarks of Leichtenstern<sup>16</sup> we are in full agreement. The essential nature (and etiology) of delirium tremens is still incompletely understood.

Before proceeding to the details of the case to be cited, I wish to mention a particularly conglomerate case which is easily classifiable by means of Southard's grouping, but quite impossible to locate satisfactorily in the twenty-two titles of the A. M.-P. A. Manual. The patient was a successful surgeon, aged 38, with a family history of multiple instances of insanity on both paternal and maternal sides. He himself had long been alcoholic and had latterly become addicted to the use of morphin, hyoscin, and, possibly, of other drugs. This man contracted influenza and developed thereafter a psychosis as bewildering as the evident etiology, combining symptoms of paranoid schizophrenia and Korsakoff's syndrome. Incidentally, his father is thought to have been paretic, and the patient himself admitted syphilis, but the spinal fluid and the blood serum proved negative.

I will now review one of our typical cases of delirium tremens. Some of these psychoses began during the acute influenza attacks, some immediately afterward, some a few days after supposed recovery from influenza.

CASE E.—*Alcohol(ism) + Influenza = Delirium Tremens.*

*Family History.*—A bartender, single, aged 32, born in Massachusetts, had a negative family history.

*Past History.*—This was negative except for the following particulars: For sixteen years, more or less, he had been alcoholic, and especially so for the past three years. Until three years ago he worked as an egg inspector at \$18 a week, but later became a saloon porter and later a barkeeper. His own conservative (?) estimate of his average consumption during the last year or two was "two gallons of whisky and beer a day." He freely admitted having been "drunk" two or three times a week for the previous two years, but denied ever having been hallucinated; and he was not a solitary drinker.

*Present Illness.*—He entered the hospital with a history of having recently had influenza, subsequent to which he became noisy, excited and destructive in reaction to hallucinations. He entered in a hyperkinetic, euphoric state and "would rather sing 'On My Way to Mandalay,' than answer questions." He was *correctly oriented*, saying he thought this would be Friday "if it don't rain," and that he was now "in an Influenza Hospital" (which it might well have been taken for at that time). He remained quite disturbed, reacting to

many hallucinations, but without great conduct disorder until the fourth day, when he suddenly became clear and gave the following account of his present illness, which is established by comparison with outside history to be correct as to known facts.

He begins by asking if he has been in a trance all this time. Says he "just woke up this morning." Is not clear about recent events; thinks it was about the first of October (two weeks ago) that he took sick with influenza. (As a matter of fact it was four days prior to admission and eight days prior to the taking of the history.) He took to bed, and on the evening of the fourth day (he thinks the second) began to imagine foolish things, heard a band in the house, thought there were robbers in the building, saw the face of a robber in the window, saw "a little coon with a cigar in his mouth and a Quaker Oats box in his hands." The "robbers" remained about, too. He became very frightened, "yelled like hell" and scared the whole household. Threw the cuspidor at "the little coon." Kept the house awake. About 7 a. m. a doctor was called and found him "standing up, all in a sweat, much excited by all that had happened." The doctor sent him here. He remembers coming, being admitted, and being visited repeatedly by the examiner. Has a hazy memory of the whole affair, including many minor details, such as discovering that he had pediculosis pubis, etc.

Recalls also numerous delusions and hallucinations of the same period, and although they were real enough to him, he strangely does not confuse fact and fiction, although he says, "I couldn't swear to it." Some of these delusions and hallucinations were that he was in a saloon where he had once worked, and that a riot was in progress, that he was reprimanded by the "boss" for slack work, that there was a parade in which he was participating, that he was drunk, that a friend named Florence was hiding under a board, that an audience surrounded him, and that "rats, snakes, mice, scorpions and every kind of an animal except an elephant" were to be seen. These he thought he saw en route to the "Influenza Hospital." This morning he woke up and wondered where he was. Finally decided that he had been "in a trance for the past week." Thinks alcohol had much to do with it, and that he had "the shakes" the day he got sick with influenza.

*Mental Examination.*—After the disappearance of the acute visual and auditory hallucinations and concomitant delusions, no psychotic symptoms were observed. He remained quite clear after the fourth day and was discharged two days later with a diagnosis of delirium tremens, recovered.

*Physical Examination.*—This was entirely negative except for general tremulousness and a blood pressure of systolic 150, diastolic 105.

*Psychologic Examination.*—This showed a mental age of 11.5 years with a variation total of 15, "excellent" performance on the memory tests and an acceptance of only two of ten suggestions.

#### GROUP 5.—*Encephalopsychoses.* Psychoses with organic brain disease

*Comment.*—"Cerebral apoplexy has been described as a symptom of influenza in England as far back as the epidemic of 1743. . . . There are no accounts of any postmortem examinations in this period. . . . In 1890 . . . I first called attention to these apoplectiform hemiplegias and monoplegias occurring in influenza. . . . Anatomically there was . . . a focal acute hemorrhagic encephalitis."<sup>34, 29, 24</sup>

34. Leichtenstern, O.: Primary Encephalitis, *Deutsch. med. Wchnschr.* 2, 1892.

Thus does Leichtenstern refer to this interesting phenomenon, influenza apoplexy which "must be regarded as a rarity." He cites cases reported, in addition to his own, by Virchow-Senator, Fuerbringer, Koenigsdorf, Schmidt, Erlenmeyer, Gross, Eichorst, Stembo, Remak, Drasche, Bilhaut, Prentis, Herzfeld, Warfvinge, Brakenridge, et al. Two cases reported by Straumann are cited which resemble the one here recorded "(fever, coma, death) . . . most readily classified under the . . . form of acute encephalitis without motor manifestations or motor paralyses." The pathology and a short discussion of the various forms is given, but he does not speak of definite psychoses accompanying or preceding the other manifestations, as herein-after related. Michell's<sup>28</sup> comment on the production of "apoplexy" by influenza has been quoted above. Oppenheim<sup>35</sup> refers in his textbook to his own work and that of Struempel, Lichtenstern and Fuerbringer, and gives a good description and photograph of the lesion. He does not discuss psychotic accompaniments.

Up to the present time the 1919 literature has contained no other reports of postinfluenzal hemiplegias (encephalitis hemorrhagica influenzae). In an address at the meeting of the Association of American Physicians at Atlantic City, June 16, 1919, Major J. W. Hall of Denver casually mentioned two cases seen by him in the course of his military medical work in the training camps of the southwest. In a personal conversation he stated that both cases were sudden hemiplegias occurring in young male adults during convalescence from influenza, one while straining at stool, and the other under circumstances not recalled. An effort is being made to collect details on these exceedingly valuable cases.

A few other instances of encephalopathic psychoses with an associated influenza were observed in our series, including one other instance of cerebral hemorrhage, but in a case psychiatrically diagnosed manic-depressive psychosis with good symptomatic basis.

It may be demonstrated later that encephalitis lethargica should be placed in this group of encephalopathies resulting from influenza. At the present time opinion on this point is sharply divided, and in order not to confuse the present issue, encephalitis lethargica (as opposed to encephalitis hemorrhagica) is not discussed or illustrated here.

*CASE F.—Normality + Influenza = Atypical Psychosis, Proceeding to Death from Cerebral Hemorrhages (Hemorrhagic Encephalitis).*

*Family History.*—The patient was a woman, aged 43, married, born in Italy. She had lived in the United States eighteen years and did housework. The history of her grandparents was negative. The father died of pneumonia at

35. Oppenheim, H.: Textbook of Nervous Diseases, translated by Bruce, London, 1911, p. 825.

73; the mother at 65 of an unknown cause. A paternal uncle died of heart trouble and his sister suddenly of unknown cause. A maternal brother killed himself while drunk (?). There are four siblings living and well, none dead. No mental, nervous or epileptoid disease was acknowledged.

*Past History.*—The woman had been taught in Italy to read and write, but little more. She was always a healthy child and was never in a hospital. She was married at 27 to a man of 46, and the union was happy and fruitful. There are nine children living and well; one died of acute indigestion. There were no miscarriages, no known mental defect in the children.

She never did other than housework. Her personality is described as being "quiet, not inclined toward recreation, but prefers to stay at home. Good-natured, and a good housekeeper. Normal interest in her (Catholic) religion." She was not alcoholic.

*Present Illness.*—Until September 10 the patient was considered well in every respect. On that date she went to bed with an acute attack of influenza. There is some question as to the duration of this disease. She is known to have been febrile for two succeeding days. She then tried to get up, but had "a spell" of an hour's duration during which she was tremulous, "nervous," suffered from palpitation, and had a fear of death. That night she was unable to sleep for fear, and this symptom persisted. She was taken to a general hospital and spent fifteen days there, during which time she was constantly agitated and querulous, complaining of multitudinous vague pains in stomach, head, etc. She went home and remained in bed for two weeks; then she was up and about for some six weeks more before being brought to this hospital. She was thought by her relatives to exaggerate trivial symptoms; she complained constantly, believing her digestion impeded, that she was hopelessly constipated, and that she could not possibly live. She made many pretended attempts at suicide and finally one confessed bona fide attempt.

There were no avowed hallucinations, no observed fainting attacks and no convulsions; and there was no delirium, no known amnesia, no fibrillation, etc. It was about ten weeks after the onset of the influenza that she was brought to this hospital.

On the day of admission, when she tried to cut her wrist vessels, "she was excited for the first time, and pushed her husband away and did not want to see any one." The admission notes at this hospital, condensed, read as follows:

"Patient is an agitated and apprehensive Italian woman who answers questions promptly and accurately. Since influenza in September . . . she has been depressed, afraid she would die, worried over her physical condition. 'I feel sick all time. I think I die. If I shut my eyes I see bad people with big eyes. If I go to sleep I wake up and worry and think never get well. It all is come for my stomach. If I eat I feel bad; if I don't eat my stomach empty, I feel better. My nerves are all excited and my nights are terrible.' She complains of palpitation, constipation, and insomnia in addition. No conduct disorder, and no frank evidence of depression."

The ward notes made thereafter state that no change in general demeanor occurred.

*Mental Examination.*—Appearance and behavior were as given above. Her orientation was precise. Her memory was not defective either for recent or remote events. She gave the full details of her family and past history, as

well as of the present illness in accordance with the outside history as related. In regard to *hallucinations*, she declared that when she closed her eyes at night she saw the faces of dead people and others. This frightened her more than ever, but the visions disappeared when she opened her eyes. The visions did not speak to her—there were no auditory hallucinations. *Ideas and Judgment*: Delusionary formation and lack of insight may be noted from the above account. Her *thought processes* showed no manifest abnormalities of attention, associations, or train of thought. Retardation was not present. "She speaks in a rather melancholic tone, whispering at times. *Emotionally*, she is more apprehensive than depressed, and greatly worried. At times she smiles pleasantly, but there is no schizothymia." Her *motor status* showed mild hypokinesia with no further conduct disorder.

*Physical Examination* entirely negative, except that the knee jerks were not obtained. Blood pressure, systolic 118, diastolic 70.

*Laboratory Findings*.—Urine, blood serum Wassermann test and spinal fluid examination, including Wassermann test, negative.

*Course*.—It is interesting to note that on the fifth day after admission the staff made the following vote on the diagnosis: Psychoneurosis, 2; manic-depressive psychosis, depressed phase, 1; undiagnosed psychosis, 4.

On the 19th, six days after admission, she was found to have a pulse of 140, a temperature of 104, and was apparently unconscious. In this condition she remained for over two days and died.

A necropsy was performed by the assistant pathologist to the Massachusetts Commission on Mental Disease, Dr. Myrtelle M. Canavan, and the brain in toto and in section was examined by the pathologist, Dr. E. E. Southard. The chief findings were: An extensive cerebral hemorrhage of large size with evidence of many petechial hemorrhages throughout the cortex, and bloody fluid in the third ventricle. There were numerous "flea-bitelike dots" of blood between which the tissue was softened and of a gray to grayish-red color, as described by Leichtenstern. In addition, there was chronic fibrous endocarditis of left auricle and of mitral and aortic valves, old pleuritis, purulent bronchitis, gallstones in duct, slight vascular nephritis, aortic sclerosis, petechial gastric hemorrhages, hemorrhagic uterine lining, acute leptomeningitis.

#### GROUP 6.—*Somatopsychoses*. Psychoses with somatic disease.

Delirium in all its polychromatic variety may be proposed as the representative symptom of this group. In so declaring we elude the pregnant question of just what delirium really is. In the paper previously mentioned<sup>24</sup> I have made a plea for the readoption of the simplest of classifications for delirium, namely, one based on the time of onset with respect to the fever, of which it may, indeed, be entirely independent. Thus all deliria associated with infectious diseases may be designated as either prefebrile (cum) febrile, or postfebrile, and in such a designation there is the added value of a prognostic index. The postfebrile forms are proverbially difficult of diagnosis and grave in prognosis. The fourth form, afebrile delirium, does not enter into the present discussion.

The fact that no form of mental disturbance is more familiar than what is termed "febrile delirium" is far from equivalent to saying that

it is the best understood. The descriptions of its essential nature are widely variant; even now there is no agreement on its pathognomonic signs or symptoms, and many—the writer included—are inclined toward agnosticism in point of pathognomonic indices, and to doubt if any such exist. Part of our difficulty herein may be referred to the psychologists, who have not yet decided just what we should believe as to the nature of consciousness; but perhaps an even greater blame falls on psychiatrists themselves for the confusion arising from the use of inaccurate, ambiguous and indefinite terminology and nomenclature.

Of the various forms of transient mental disturbance whose general character fitted the hazy outlines of the psychosis "delirium," certain types were particularly striking. The "muttering delirium" of historic fame, the "typhoidal delirium," the acutely maniacal forms of delirium with homicidal tendencies, types simulating Korsakoff's syndrome, delirium without disorientation, and divers others were encountered. Two types deserve special mention. In not a few cases there was a history of wandering about the neighborhood unable to find the way home, pounding on the doors of houses in alarm or terror, disappearing from work or home and later discovered by the police, amnesic and disoriented. These usually occurred during convalescence. An informal and unofficial report from the medical officers of a certain naval prison is to the effect that a number of the prisoners provisionally classed as "deserters" absconded during the convalescent period following influenza, and it was found that some of these, at least, were amnesic for events of the period and represented, in the minds of the naval medical officers, additional instances of postinfluenzal twilight states.

A second unusual form of delirium is what might be denoted delirium schizoprenoides, the schizophrenic elements dominating the picture to such an extent that the differentiation from dementia praecox in the cross section was quite impossible. This type seems to have been exceedingly frequent after the recent epidemic, as it is mentioned in practically all of the reports on mental sequelae of influenza which have appeared. (Burr,<sup>27</sup> Egbert Fell,<sup>36</sup> Harris<sup>37</sup> and others.) Because of the frequency of this type in our series, and the remarkable configuration of the clinical picture, it is this type of which I have chosen to give an illustration. The following may be considered a representative example of this type.

36. Fell, Egbert W.: Postinfluenzal Psychoses, *J. A. M. A.* **72**:1658 (June 7) 1919.

37. Harris, A. F.: Influenza as a Factor in Precipitating Latent Psychoses and Initiating Psychoses, with a Brief History of the Disease and Analysis of Cases, *Boston M. & S. J.* **180**:610 (May 29) 1919.

CASE G.—*Normality (?) + Influenza = Delirium*; first maniacal, then schizophrenic, then depressed, then stupid; finally disappearing.

*Family History.*—The patient was a woman, aged 34, a Nova Scotian, married and had always done housework.

The paternal grandfather died at 72 of pneumonia. He was a Nova Scotian fisherman. The paternal grandmother, a "kind, sociable, Christian woman," died at 93 of pneumonia. The maternal grandfather died at 72 of Bright's disease; the maternal grandmother, at 64 of a tuberculosis which she was known to have had many years. Her father was a carpenter 62 years old, a church deacon, said to have cancer of the lip. Her mother, the chief informant, seemed to be intelligent and well informed. She thinks she has had heart and kidney disease for some ten years. The uncles were chiefly sea-faring men; those who were not drowned are in general still alive and well. A maternal sibling died of tuberculosis of the brain; a paternal sibling of consumption. Aunts numerous, nothing of importance. Siblings: Brother, 37, well; the patient, 33; sister 31, with two healthy children; sister, 29, with three healthy children and one dead from tuberculosis; sister, 19, died of tuberculosis; sister died of pneumonia at 3; sister, 22, well. No known mental or nervous disease in any branch of the family.

*Past History.*—The patient was born on the Nova Scotian coast in 1886, and lived there until the age of 22, when she came to Boston and has lived here since. Educational: She attended the grammar and high schools from the age of 6 to 17, with a good record throughout. Habits: No alcohol nor tobacco; her meals were regular, and her exercise fairly regularly.

*Personality:* "Active, lively, talkative, high spirited, kind, practical, independent. Fond of music, is much interested in the Eastern Star fraternity. Member of Baptist church." Later we were told, however, that she had always been extremely suspicious. Marital: She had been married four years prior to her present illness. Her husband was a rather egocentric individual, "demanding more than he gave." The union was happy, however. A child, born less than a year after marriage, is well and strong, although he had influenza and pneumonia along with his parents. Medical: Except for the diseases of childhood, the patient had no serious nor protracted illnesses except typhoid fever, which she had at the age of 14.

*Present Illness.*—Husband and wife were taken ill with influenza about November 17. On November 24 the husband died. Even before his death, the patient began to show definite evidences of psychosis. "The room was filled with the Holy Ghost." "The spirit of God" told her he would not die. She kept saying, "Don't you feel the stillness?" and made the family pray with her constantly.

The day after her husband's death she was up and out of the house, seemed fatigued, talked less, and was less religious. The following day she spoke of having died, gone to heaven and returned; insisted that she was God, and wished to convert everyone. The following day she went with her sister to attend to some business. She seemed alternately dreamy, deluded, semi-conscious, and in a state of complete clarity. On the evening of the latter day she showed evidences of great fatigue, "continued to scream, laugh, sing hymns, and wanted others to do so." This continued for the next day or so. She then began to claim supernatural power, talked of striking people dead or blind, insisted that she was God, and her child the devil. Because of her threat to show what she would do to "the devil" she was brought to this hospital lest she injure her baby. She was brought by the police.

*Mental Examination.*—On the day of admission the patient manifested great excitement. She was constantly talking, shouting, or singing. A sample of her discourse at this time follows:

"Now I wish you to know that at this present time I am God. I am God. I am God. You understand, I am God. God says, 'Now let us sit down and reason together. Laugh and the world laughs with you.' Weep, now Father, I don't want to do that. God so loved the world that he gave," etc. (Where does that come from?) "The Book of God, do you know that?" (Then sang one verse of *The Light of the World Is Jesus*.)

(Why do you assume the personality of God?) "Write down for me the word G-O-D, God. You are fully convinced that I can see who I am, are you? Before I get through with you I will convince you that you don't know who you are, but I know who I am. I am M-M-M, and God is speaking through me. No, I am a scientist, I am only a Christian girl. The Blessed Virgin is right there with God, yes, she talks to me too. I can see them both either in daylight or at night. I have a mission on earth, it is to bury my husband, N-N-N."

*At this time she was correctly oriented as to time, place, and, nominally, for person.* Her replies to questions, while frequently irrelevant and incoherent, indicated that her *memory* for recent events was "not appreciably impaired." Her *ideation* showed expansive, delusionary formation, centering about a transformation of personality. Her *insight* did not extend beyond declaring that she was "a religious fanatic." Her *thought processes* showed unstable attention, loose and incoherent associations, and an accelerated train of thought without true flight of ideas. Irrelevancy in her replies was less common than incoherency in the statements themselves. At times she became obscene and profane. Her *emotional tone* was one of excitement, expansiveness, agitation without apprehension or elation. Her *motor status* was that of hyperkinesis in rather narrow limits. She was exceedingly untidy and kept her room and her clothes soiled.

This description is representative for the first five days of her stay. Thereafter the picture changed, as will be detailed below. The record of the following conversation was made on the fourth day toward evening, when she was rather less disturbed than usual. Except for the queries indicated in the parentheses the matter was entirely spontaneous.

She is in a private room, constantly jabbering and shouting, running about the room, crouching in a corner, or gesticulating. A stench of fecal and other origin fills the room. Her gown is soiled with a menstrual discharge, on which fact she comments unabashed. When the examiner opens the door she forces her way past him and leaps on a bed in the corridor. Here she perches immovable, silent for a few minutes at a time, but bursting out at frequent intervals in a senseless and incoherent comment on some word or phrase caught from an adjacent patient who is in a pack and much disturbed. (What are you doing?) "Why, I am rowing a boat. I am on to Bermuda. What you writing? (Snatches notebook.) Why don't you go and get me a glass of water? There, there, that's what it is. I am telling you what the nurses are doing. I will now convince you that I am not blind. You know damn well that I am going to unbraid my hair. If I get there. . . . You know damn well that . . . here . . . there . . . (her loosened hair falls over the bed railing). This is the fountain of life. Don't you touch that or I'll drive you through the wall so quick you won't know whether you are a bird or a nurse! Why, I told them you got all those supreme beings and the birds

and. . . . See! See! That's what they do! Isn't it terrible! (Jumps on bed and assumes threatening attitude, but is quieted by nurse.) Get out of my way every damn one of you! Don't you dare to take that out of my mother's lips tonight, and I think it's a damn shame that I am still without that glass of water. You damn nurse I'll slap your face in a minute! You'll find your birds on that boat Bermuda, and I'm still without the glass of water. I want a drink, you damn fool. (Overhears the word "skeletons.") I'll tell you who will be skeletons around here; the doctors and nurses. I'll poison every one of you. And I'm still without that glass of water. P, I, S, S, there, I'll call you a damn sweet doctor." (Nurse walks by.) Nurse says, "Excuse me." Patient replies, "Oh, you damn fool! (mimics) 'Excuse me, excuse me, excuse me,' what's that for?" (Water is brought. She obeys orders to keep her hands down and drink from the cup held by the nurse. Stops between draughts to say—) "And I'm still sitting here trying to get a drink of ice cold water. (Spits on the floor.) Now you are fully convinced that I did not have that glass of ice-cold water. (Supper is brought. She asks for sugar, is told that there is none, says all right she will drink the tea without it. Examiner remarks that she is a "great girl.") "You bet I am. That's the trouble, it's mind over matter. (Bursts into tears.) I'm crying to think I was treated so for calling you the sweet-faced doctor." (She hears another patient singing, and herself breaks into song, but in another key and tune.)

*Physical Examination.*—At this time the physical examination was essentially negative; the urine negative; the spinal fluid negative; Wassermann reactions on blood serum and spinal fluid negative.

*Course.*—The excitement gave way to a phase of inert apathy. She wandered about the ward, quiet and well behaved. She obeyed orders, retained her clothes, associated with other patients, but was rather noncommunicative, played the victrola, and was rarely or never talkative after the tenth day. On the fourteenth day irrelevancy, incoherency, inadequacy characterized her replies. Thus:

(What does God say to you?) "What's wrong? Nothing's the matter."

(What is the trouble with that girl?) (indicating a patient.) "I can only answer for one."

(Are you clear in the head now?) No reply. She stares inquiringly; glances furtively about.

(How do you feel today?) "It was, yes sir. Better."

(Are you better?) (After a pause) "My breath is very offensive."

On the sixteenth day she was quite disturbed in the forenoon, but toward evening became more composed. Cried when her husband's death was mentioned. Answers still illogical, irrelevant, incoherent. No insight. She talked a little of having been "a religious fanatic," but the words seemed to have no real meaning for her.

On the eighteenth day she was interviewed in continuous baths and was quiet and accessible. (How long have you been here?) "You know me as well as I know myself."

(What is that in your hand?) "My handkerchief."

(Why have you wadded it all up so?) "Why mother gave it to me."

(Why are you in the bath?) "She said it was necessary" (pointing to the nurse).

(Are you suspicious, Lillian?) "Of you?"

(Of any one?) "No." (Pause, looks at nurse) "But I know she don't dare wink any more."

(What?) "I said she wouldn't pull my hair again."

(Did she do so once?) "Ask her. Let her speak for herself. 'Speak for yourself, John.'—Miles Standish."

During the next week the patient seemed more and more quasi-normal, the chief symptoms being restlessness and the occasional expression of paranoid ideas. Irrelevancy and incoherence in her replies disappeared almost completely. A certain tendency to evasiveness was noted. About this time she was asked to write her version of her trouble, and wrote the following account:

"I was suffering from severe headaches for over a week and had been without sleep for one week previous to being brought here. I was so disturbed in my mind about where my husband's soul would go that I prayed with my Maker and asked for His help in my afflictions. I felt my prayers were being answered and I felt the presence of God around me. All people would say was 'sleep.' I became somewhat of a religious fanatic and heard voices speaking inwardly. I quoted passages of scripture and the Twenty-Third Psalm ran through my mind. I talked to those around me and spoke of the teachings of learned men that I had heard preach from the Bible. One day, in taking my baths, I spoke of Billy Sunday and told one of the patients about it. I was somewhat emphatic in assuming so much Godlike spirit, but my nerves were somewhat unstrung, and while I still feel the need of prayer I am able to control my nerves and know that I have been helped in my treatment here. It all came from my mind being overtaxed by thinking so steadily. But thanks to all around I feel my feet are on terra firma once more and I realize how disturbed I was."

*Condition on the Thirtieth Day.*—The patient had been transferred to the quiet ward and was daily employed in ward work and occupational therapy. She seemed, superficially, quite normal, with partial insight as to her recent mental trouble. She recalled many of the incidents, but was unable to recall many others. She claimed to have regained entirely her old interests and affects and was complacently waiting to be discharged.

Certain residual symptoms remained, however. A nurse's note is quite expressive. "Patient depressed at times. Cries easily, but says she is strong enough to go home. Seems suspicious. Thinks conversation of others is directed toward her. Asks the same question several times."

Moreover, she seemed somewhat slow in grasping the situation. She doubted if she were really to go home as had been announced to her and cried at the realization that it was true. She lingered about the ward office as if fearing something might be said about her. Her psychometric rating, moreover, gave her a mental age of only 13—on Point scale;  $13\frac{7}{12}$  on Stanford.

She was discharged on the thirty-first day, virtually, although not technically, against advice, improved but not recovered. The diagnoses varied widely, ranging from cyclothymic psychosis to dementia praecox, according to the period of consideration. On the whole the case seems to be quite representative of one of the polychromatic forms of postfebrile (influenza) delirium.

*Subsequent Report.*—Two weeks after discharge the patient reported. At this time she betrayed a peculiar paranoid state difficult to define. The following conversation is illustrative of the irrelevance, inadequateness and suspicion which characterized her manner. She insisted that she was perfectly well (as did also her mother).

(We are in doubt as to how much can be remembered by delirious patients. Will you help us? How much do you remember?) "Well, I shouldn't remember everything, should I?"

(I don't know, do you?) "Well, it should be a little hazy, shouldn't it?"

(I don't know, is it?) "Well, to a certain extent."

(To what extent?) "Well, I don't suppose I remember everything. Can you remember everything that ever happened in your life?"

(We want you to come in and do another psychologic test.) "What for?"

(I think you could do one perfectly now. You didn't before.) "Yes, I did."

(No, it wasn't so good.) "Well, let some one else do it. You do it for me."

(Did you think you were God?) "I never said that."

(Yes, you did.) "Doctor, I never said that at all."

(You admit that you don't remember everything.) "Well, I didn't mean what you mean."

(You admit that you said you were God?) "I didn't."

(But we have it written down.) "Oh well, that's different."

(But you said it, you know.) "Well, I didn't mean what you mean. You can't read my mind and I can't read your mind. No one can know what I felt unless he has been at death's door. Have you ever been at the brink? That's where I was. I had only a flicker of life. I felt my pulse—it was very slow—about 40."

(Was it then that you heard and saw God?) "I said I was the armor of God . . . I had . . . that's what I said."

(What did you see?) "I saw everything."

(Spirits?) "Yes."

(What were the spirits like?) "Doctor, don't you have a conscience? Doesn't your conscience tell you things? That's what it was."

(Did you see angels?) "Yes. They weren't like you, though, Doctor, not like you, waving around in the air."

(What were they like?) "Doctor, you can't experience it unless you have been at death's door."

(I don't want to experience it. I want to hear about it.) (Reply was inadequate—details forgotten.)

The mother seems to be an intelligent woman who appreciates the evasiveness and paranoid trend of her daughter's replies, but says that she has been that way from childhood up. "As a child she was suspicious and I had to take care lest her feelings be hurt by trivial things that didn't concern her. If I spoke quietly to one of the other children, she would suspect that I was talking about her. She is absolutely the same now as she has always been. You have to know her to understand her ways. That is her, all over."

The further course of this case will be recorded in a later article of this series dealing with delirium and its relation to schizophrenia. For the present it serves admirably as one of those baffling cases, of which we had so many, wherein there was a constant battle between the protagonists of a diagnosis of schizophrenia *paranoides* and the advocates of the simple designation delirium (*schizophrenoides*).

#### GROUP 7.—*Geriopsychoses*. Psychoses of senile and presenile periods

In a post graduate lecture in 1893 Gowers<sup>38</sup> mentions a case representative of many others wherein full mental vigor was replaced by utter dementia in a senescent individual. The influence of the acute

38. Gowers, W. R.: The Nervous Sequelae of Influenza, *Lancet* 2:1 and 73, 1893.

infections is vaguely enough understood at best, and our knowledge of the effect on the mental diseases of old age is the least favored in point of available material. The following example speaks for itself. The writer is quite conscious of the deficiencies in diagnostic data, but the known facts are strongly indicative on the basis of probabilities.

CASE H.—*Senescence + Influenza = Senile Psychosis, Paranoid Form.*

*Family History.*—A married man, aged 61, born in Maine in 1859, had a negative family history.

*Past History.*—He had had the usual children's diseases, but no severe illness nor convulsions. After a cursory education he entered the shoe business and remained at that work consistently the rest of his life. Habits: He had been a total abstainer from alcohol. Marital: Married for forty years to one wife who died of "creeping paralysis," and for two years to a wife who is living and well.

*Present Illness.*—The patient was a Christian Scientist whose orthodoxy prevented his consulting medical advice and likewise prevented our acquisition of a good account of his trouble. The following admission note gives all that was ascertained.

"Influenza began approximately October 19. Although not bedridden, he was feverish for several days. Mental symptoms appeared a few days afterward, about ten days before admission, rather insidiously. According to his wife and a friend, extreme talkativeness, restlessness, insomnia, and delusions of government inspectors pursuing his friend are the chief symptoms. In the admission office he was very indignant, having been brought by strategy, and insisted on having it explained why he was brought, what the disturbance was all about, etc. He talked at a great rate about his unblemished record, and made remarks and hints which referred to the delusions above mentioned, but denied them when flatly put to him. Absolutely no insight. He has a marked aversion to doctors and hospitals. Quite resistant on way to ward."

The physician who examined him on the ward noted: "He is a very talkative and extremely irritable man, who can give no reason for coming here, but demands that some explanation be given him. He refuses to admit anything, even that he has just had influenza. Very circumstantial and rambling in his conversation. He is apparently well oriented and his memory seems very good. No delusions or hallucinations are elicited. Very irritable. Radial arteries markedly palpable."

*Mental Examination.*—After the day of admission depression, humiliation, and chagrin replaced the irritability and disagreeableness. He was a rather pitiful sight, sitting about the ward in great dejection, and while superficially accessible would give no adequate expression of his ideation except that friends had wronged him. His thought processes showed marked circumstantiality, occasional irrelevance, and a distinct tendency to wander from the subject at hand.

*Emotional Tone.*—Depression, irritability and agitation were represented at different times. No considerable amnesia, disorientation or disturbance of motor status and no hallucinations were ascertained. No further data concerning the delusions were obtained than those mentioned in the admission note, save that he was fearful that something would happen to him here.

*Physical Examination.*—He was fairly well developed, but poorly nourished; his hearing was slightly impaired; he had a slight tremor of the tongue, normal reflexes, no teeth. His lungs showed fair resonance throughout, except for a slight dulness in the right base, numerous râles in the left back, and a reputed friction rub in the left axillary region. Heart: The apex was felt in the fifth space. The right border was 2.5 c.c. to the right of the midsternal line; the left border, 9 c.c. to the left. Aortic and pulmonary sounds were of good quality; no murmurs were heard. The pulse was equal, regular, and synchronous. Arteries (temporal and radial) were tortuous and sclerosed. There was a pulsation of the neck vessels. Numerous dilated capillaries were found on the legs. The blood pressure was from 150 to 76. The physical examination was otherwise negative.

*Laboratory Findings.*—These were entirely negative, including urine, spinal fluid, and blood Wassermann test.

*Course.*—A lumbar puncture was done on the sixth day. He was kept in bed twenty-four hours thereafter. On the eighth day he complained of a little headache and some nausea. He made no other complaints and went to bed as usual on the evening of that day. At 8:30 p. m. the nurse entered his room and spoke with him. He was quiet, and there was no complaint. He was seen at least twice during the night by attendants, and nothing unusual was noticed. At 6:30 a. m., November 9, as he had not arisen when the other patients arose, the nurse went to call him, entered his room, and found him dead.

The medical examiner was notified and inspected the body before its removal from the bed or the room. He expressed the opinion that death was due to cardiovascular disease and did not recommend a necropsy. Whether or not this diagnosis by inspection postmortem resulted in a correct physical diagnosis, the fact remains that the psychiatric diagnosis of psychosis with cerebral arteriosclerosis is not tenable. Neither the amnesia nor any evidences of focal or general cortical irritation impairment, or destruction, were present. On the other hand, the vague persecutory delusions, the total absence of insight, the irritable, depressed and agitated emotional tones, plus the history of insomnia and restlessness, together with the age of the man, make a diagnosis of senile psychosis of a paranoid form the most reasonable hypothesis.

#### GROUP 8.—*Schizophrenoses. Dementia praecox*

The conception of schizophrenia as delimited by Kraepelin<sup>39</sup> and elaborated by Bleuler,<sup>40</sup> was not in the psychiatric mind at the time of the last great influenza epidemic. Consequently, there is not in the literature any elaborate discussion of the rôle of influenza in the production of the picture of dementia praecox. And, as stated in the introduction, no other acute febrile disease occurs in sufficient ubiquity and sufficient numbers to make possible accurate comparative study of its psychic effects under uniform extraneous conditions.

That influenza may be followed by schizophrenia is mentioned directly in a few instances, and indirectly rather often. Thus, Sir William Gowers<sup>40</sup> wrote in 1893: "Just as the depression develops

39. Kraepelin, E.: *Psychiatrie*, Ed. 8.

40. Bleuler: In Aschaffenberg's *Handbuch*, Leipzig and Vienna, 1912.

into melancholia, so the delirium which occasionally attends the acute affections may have for its special sequel chronic delusional insanity (read dementia praecox) and, very rarely, acute mania." Again, without entering into a discussion of the proper distribution of the entities composing the heterogeneous, ill-defined and fortunately obsolescent syndrome "amentia," it will be recalled that Kraepelin pointed out that many cases so diagnosticated proved to be dementia praecox, and that Régis<sup>41</sup> regarded a prolongation of "Confusion Mentale" (essentially the same concept) as practically identical with one of his two forms of dementia praecox (constitutional and incidental). And as "amentia" and "mental confusion," etc., are frequently mentioned as sequelae of influenza in the older literature (including Régis' textbook), one may presume that the occurrence of dementia praecox after influenza, although frequent, by a confusion of nomenclature escaped signalization.

The few authors who mention it specifically do so with an apologetic manner, generally ascribing its occurrence to a coincidence. Kirn, Bonhoeffer and others mentioned above have referred to it, but generally add reassurance that definite stigmata of psychotic tendencies were previously manifest, or were apparent in the family history. Paton<sup>42</sup> remarks the occasional precipitation of Schizophrenia by influenza, but ascribes to it only a minor rôle.

These writers were in general still strongly influenced by the functional conception of Schizophrenia. The great majority of them were primarily clinicians. And because of the greater availability of material, the protagonists of the organic basis have attacked the problem almost altogether from the pathologic side. In the specific instance of influenza, Gosline<sup>22</sup> reported a series of necropsies with histologic brain findings, and pointed out the similarity of findings in a case of influenza with delirium, and cases of dementia praecox, "drawing the obvious inference, "that . . . certain cases of dementia praecox are due to infectious or toxic processes."

Perhaps the organicists have overlooked what might seem to give considerable weight to their thesis, accruing from the clinical side, as observed in the recent epidemic, and in the present series. In the statistical study previously mentioned<sup>24</sup> I pointed out the surprisingly great numerical incidence of schizophrenia, and concluded that "an organic basis for some instances of the picture denoted dementia praecox is supported by" this and other facts of that series.

41. Régis: *Psychiatrie*, Paris, and, *Délire de la convalescence*, Ann. méd.-psych., Paris, 1883, p. 393.

42. Paton, S.: *Psychiatry*, Philadelphia, J. B. Lippincott Co., 1905.

I now propose to present typical instances of this phenomenon.

Because of the emphasis placed on the matter of predisposition by most writers, two types of cases are recorded. The first two with a history satisfactorily demonstrating a tendency toward the seclusive and egocentric; and the third one clearly without any such stigma. As cases similar to both of these types were, as previously reported, by all means the most common forms of mental disease, the choice is rather wide. Many of the cases, however, have minor complicating factors: the history of a recent miscarriage; an incomplete record of the past history; defective laboratory reports, etc. In general, the cases are quite representative and fairly complete. Two instances of the precipitation of second attacks of the cyclic form of schizophrenia occurred. These are not here illustrated.

*CASE I.—Predisposition (egocentric temperament) + Influenza = Full blown Schizophrenia.*

*Family History.*—An unmarried nurse, aged 35, was born in Massachusetts. Her grandparents were born in Ireland, of whom there is no history. Her parents also were born in Ireland. Her father died at 65 with pneumonia, the mother, at 67, of shock. There were paternal and maternal siblings, of whom there is no history. The patient's siblings: two sisters, living and well; one brother aged 38; a twin sister died of gallstones. A nephew died of tuberculosis. No history of mental or nervous diseases in the family.

*Past History.*—The patient was born in a Massachusetts village. She graduated from grammar school. Not much is known of her childhood. At 26 she graduated from a nurse's training course and since that time has been a visiting nurse for the Boston Consumptive Hospital. *Medical:* The patient suffered a Colles' fracture five years previously and was described as having had "neuritis" for a period of seven months three years ago, which was connected in some way with her diet. "A stomach trouble" which was suspected by her friends to have been neurotic, had been complained of for many years. *Habits:* She took no alcohol whatever. She paid much attention to treating herself and lived on a special diet because of this "stomach trouble." Her work gave her a reasonable amount of exercise.

*Personality.*—The patient is described by the superintendent of nurses, who had known her for eight years, and who seemed to be an intelligent, reliable and observing person.

"She talks continually about herself. She has always been egotistical, selfish, self-satisfied. She is not popular, but she continually talks about herself and she was always looking for sympathy. No matter what subject is brought up she will invariably bring it back to her own illnesses. She has a few friends, but is definitely seclusive and does not go about with them much. She is loud and noisy in her actions, speech and laughter, and her laugh is empty, forced and hysterical. She poses as younger than she actually is and never realizes her age. She is either way up or way down. She is stingy with money, except for herself, and spends much on dress. She is a devout Roman Catholic."

*Present Illness.*—She contracted influenza on September 28. She had a high fever which disappeared on the fifth day. It is thought to have been as

high as 104 F. and known to have been 102, but she was at no time delirious. On the third day she seemed quite hilarious, saying that she had the influenza and might as well make the best of it. The next few days "she began to worry about her treatment as usual."

By the time her temperature had returned to normal, she was distinctly psychotic. She is thus described: "First she would be rigid and staring and would not speak. She would relax if grasped by the arm. At other times she would talk a blue streak. This was not irrelevant or incoherent, and she could tell the doctor accurately what happened during the previous twenty-four hours. She paid little attention to people or to things happening in the room."

On the ninth day she thought she was going to die and was anointed by a priest. She insisted that she still had influenza and said that she had been accursed and told people not to come near her on that account. She slept little or none. At 3 a. m. on the tenth day she became violent, disordered her room and hurled objects at any one entering. She finally quieted down, but became excited and disturbed again the following day. Then she was seen by a reliable physician, who examined her carefully and concluded that, in spite of very active reflexes, she did not have meningitis which he had suspected because of an apparent stiffness of the neck. She was deaf for one day. She continued to show major and minor conduct disorder, but without any loss of memory or orientation, and without hallucinations, suicidal threats or homicidal ideas. Delusions were very vague. She would make such incoherent remarks as, "I have picked out a rat hole," and "I am a slacker and I am not going to die." She was sent to this hospital on the eleventh day.

*Mental Examination.*—The patient was never fully accessible. For the first few days she answered questions, but with varying degrees of relevancy. Sometimes she would make sensible and accurate replies to question, but at other times she would refuse to answer or would reply with irrelevance, nonsense or inadequacy. After the first few days she became almost completely inaccessible. She mumbled frequently to herself about being cursed, going to hell, etc. She whispered to herself, attitudinized, gesticulated dumbly, answered hallucinatory voices, occasionally jumped impulsively from her bed and walked to one corner of the room, and returned to bed without a word, repeating the process for each corner of the room. She admitted hallucinations of God's voice, and those of various persons.

Subsequently, she lay almost motionless in bed, completely inaccessible, almost completely mute. Her face wore at times a pained, worried expression, but in general it was masklike and utterly expressionless. She showed negativism, catalepsy, and on a few occasions a transient *flexibilitas cerea*. She occasionally answered questions to the extent of complaining that her mind was being influenced, that people will not let her talk, that she was dead, etc.

One or two days during the second month she would answer a few questions relevantly and correctly, but with an utter indifference and with a far-away stare and absorbed manner which is difficult to describe, but which is perfectly familiar to any one with psychiatric experience.

At the present moment, three months from date of admission, she remains precisely the same. Occasionally she bursts into a mumbled jargon of incoherences accompanied by a silly, meaningless smile; at other times she renders a loud, wild shriek or moan without any apparent cause whatever. But in the main she lies stolidly in bed, making no motion, speaking no word. Catheterization and tube feeding have occasionally been necessary.

Physical examination and laboratory findings were negative.

*Diagnosis.*—Catatonic schizophrenia (dementia praecox).

Another case further illustrating the same paradigm, but in the male sex, follows in abridged form.

CASE J.—*Predisposition (seclusive temperament) + Influenza = Schizophrenia.*

*Family History.*—The patient was a soldier, aged 27, single, and gave a negative family history.

*Past History.*—He was born in Vermont in 1891. His past history is not important. In temperament and disposition he is described by his brother as having been always of a quiet, seclusive disposition, never mixing with others, but preferring to remain alone. He was considered normal mentally by his employers, friends, and family, and he was not regarded as eccentric.

*Present Illness.*—He developed influenza and pneumonia at Camp Devens where he was a private in the infantry. He was delirious and did not regain his mental faculties. He knew his mother and father except on their last visit to him, at which time he had evidently recovered from everything except a mental disorder. His brother saw him two weeks ago and was recognized by him then. An interested friend writes: "I am informed that when he took sick he was out doing trench work, was missed at roll-call, but was not found until next morning, lying in the trench where he had been working after having lain out all night in a cold rain."

The military authorities sent only the information that subsequent to the influenza "he has been in a catatonic stupor; eats little; unclean in his habits; absolutely mute and unresponsive to external stimuli." He had been in the psychopathic ward of the base hospital for two months.

*Mental Examination.*—He was never accessible. He lay passively and apathetically in bed, responding to no questions by look or word. He obeyed simple orders, however, and cooperated in a fair way in the neurological examination. He made very mildly resistive maneuvers at times. After the first few days he was up and about the ward and was seen to look through magazines. He continued to show hypobulia and complete apathy. When offered a hand, he presented his own, and shook hands listlessly. He could not be urged, persuaded or forced to speak, smile, laugh, or cry. His facial expression remained fixed and impassive and his thought processes showed complete blocking. He seemed at no time unaware of his environment; his reception of external stimuli was not interfered with. Thus, when told to indicate by signs his interpretation of some object, he did so slowly and disinterestedly, but quite correctly.

*Physical Examination.*—This was negative, as also the laboratory findings, including spinal fluid, and Wassermann tests were entirely negative.

CASE K.—*Normality (1) + Influenza = Schizophrenia.*

*Family History.*—A single woman, aged 20, was a bookkeeper by occupation and was born in Massachusetts. Her grandparents' history was negative. Parents: Father, 42, salesman; temperate user of alcohol; well. Mother, 41, always strong and well. Although described as being of a nervous temperament she was cheerful and not seclusive or sensitive. She had a premature menopause at 28, at which time she had "hysterical attacks." In these attacks she became excited, exhibited mild conduct disorder, and sometimes fell to the floor

with flushed face and some frothing, "but never lost consciousness, bit her tongue, injured herself" or passed urine or feces. Since the abatement of attacks she has grown entirely strong and much less "nervous" and "now has good self-control," and a cheerful, even temperament.

*Past History.*—The patient was born in Chelsea, in 1898; normal but somewhat difficult delivery. She was developed normally, physically and mentally. Puberty occurred at 13 years; no disturbances. Education: She started to school at 5, quit at 16, two years of high school, one double promotion at 11 or 12. Never held back. Did well; mathematics difficult. Very quick in languages. Never held back or kept out. Economic: From 16 to 18 she was home with her mother. At 18, she became a bookkeeper at \$12 a week. Has worked steadily with little vacation for the same people; eight-hour day but little responsibility until last three months. No court record or marital history.

*Personality.*—Lively, talkative, systematic, practical, level headed, social "but does not care particularly for social functions"; sympathetic, generous, sociable at home and domestic. Goes to theater with her mother. Plays piano. Has many friends. Pleasant, even disposition. Not easily influenced." (Given by father and mother who seemed to be intelligent and honest informants.) Religion: Protestant, little interest. Habits: No alcohol. Meals, wholesome and regular. Does not care for men. Thrifty, and a good housekeeper. Medical: Whooping cough at 6 months. At 1 year, measles (slight). At 18 months, tonsillitis. Has never been ill or had a physician since. No nervous attacks.

*Present Illness.*—"Patient is very ambitious and since the head of her department went into the army in June, she has had great responsibility. Has had charge of payroll of 150 people, directed the bookkeeping and practically the entire office. Worked very hard and loved it."

September 28 patient went to bed with influenza. She was febrile for ten days, but not delirious. On the twelfth day a neighbor who is described as being a "religious fanatic" called on the patient and the parents ascribe to this interview a psychogenetic influence which it probably does not deserve. However, that evening the patient was very depressed, wept, and said she knew she was going to die. As a matter of fact she was much better. Three days later the family moved, although the patient was still bedridden. She continued to improve. However, four days later (nineteen days after the onset of the influenza) she became mute.

"She has not said a word to her father since then, although she had what seemed to be normal periods. Her mother said that she appeared more herself in the morning when she had had a good night than later in the day when she had grown tired. A menstrual period due on the nineteenth was missed for the first time in her life. She grew steadily worse; more quiet, apathetic, and showing minor conduct disorders. On five occasions she wanted to undress at improper times and places. Twice without apparent reason she went out in her nightdress. Occasionally she emitted bursts of laughter or weeping without adequate cause." She was brought to the hospital on November 6, still completely mute.

*Mental Examination.*—A fair, comely girl, rather slight, though well nourished with a face of intelligence and culture, but entirely devoid of expression. She practically never spoke. All queries were met only with a Mona Lisa smile, and no indication that they were heard. Once after persistent questioning she showed some annoyance and ejaculated, "Damn!" at one other time she said two or three incoherent words. Occasionally smiled. Otherwise there was no

response whatever at any time. A nurse's note has it that she occasionally spoke to them and that she sang a few times while in the continuous baths.

*Motor Status.*—She was quite resistive to any examination, and gynecological was impossible. She ate and slept fairly well, but showed a distant aboulia, sitting about quietly without any show of interest. Frequently she would suddenly jump from her chair, run toward one of the physicians on the ward, and just before reaching him stop short, giggle in a wild and strange manner, and walk quickly back to her seat.

*Emotional Tone.*—She showed complete apathy. Occasionally there was some irritability; generally she wore a pleased expression and in a few days she seemed to be mildly interested in her environment, but in general she was quite indifferent.

*Physical Examination.*—This was entirely negative except for hyperactive knee jerks.

*Laboratory Findings.*—These were entirely negative, including spinal fluid and Wassermann.

*Diagnosis.*—She was committed after two weeks with a diagnosis of schizophrenia.

#### GROUP 9.—*Cyclothymoses.* Manic-depressive psychoses

Gowers' dictum on the frequency of "melancholia" and the rarity of "mania" after influenza has already been mentioned, and as it is a representative conclusion of the psychiatrists who wrote concerning the 1890 epidemic, no others will be quoted here. It is an amusing commentary on the older diagnoses, that in a recent examination of the records of a state hospital for the years succeeding 1890, the diagnosis "melancholia" was almost universal on all cases suspected of having had grippe. This in spite of quite good clinical descriptions which clearly made out cases to be schizophrenic, maniacal, delirious, etc. I have previously pointed out<sup>24</sup> that in psychiatric subjects depression is distinctly one of the less common symptoms. Manic-depressive psychosis appears, however, in episodes usually of the manic form, with considerable frequency in our series, in contradistinction to the reports of Gowers and others, and possibly due to more than mere differences in nomenclature and nosology.

Under this group three cases are here reported. In the cyclothymoses (or as I would prefer to denote them, the cyclopsychoses) there are, as with the preceding group, the individuals who have manifested a cyclothymic tendency either by attacks or by disposition, and those in whom no such history appeared. On this basis the cases given below were selected, to illustrate

CASE L.—Cyclothymic psychosis, manic phase, previous attack.

CASE M.—Cyclothymic psychosis, manic phase, no previous attack.

CASE N.—Cyclothymic psychosis, depressed phase, no previous attack.

(An instance of the occurrence of the depressed phase in second or third attacks could be added, but the analogy to Case L is so obvious that it is omitted to save space. Only a few such occurred.)

**CASE L.—Cyclothymic Tendency** (Previous attack of psychotic depression) + *Influenza* = *Hypomania* (Cyclothymic psychosis, manic phase).

**Family History.**—The patient was a man, aged 22, Jew, born in the United States of Russian parents. His paternal grandparents both died of pneumonia; maternal grandparents "of old age." Paternal uncles living, three; dead by trauma, one. Maternal side: one uncle insane, "was quiet and would not speak," died at a Massachusetts state hospital at 38 after three years' residence. Father, aged 50, living and well. Mother, aged 45, said to have diabetes.

Only the one instance of mental disease in the family history.

**Past History.**—Born in Massachusetts; negative history of birth, infancy and childhood, except for enuresis until 14 years old. Attended school from 4 to 20, taking part of the high school work, including the commercial course. Was obliged to repeat the first, second and sixth grades. Deportment always good.

**Occupation.**—Left high school to go to work as a clerk in his father's store. Joined the army August, 1918.

**Medical.**—Diphtheria and smallpox in youth. Gonorrheal infection at 20. Herniotomy a short time prior to this.

**Habits.**—Total abstainer from alcohol. Masturbation from 6 to 12, and sexual promiscuity from 7 till the present time. "Cabarets and vaudeville have been his chief forms of amusement."

**Disposition.**—Very happy, sunny, constantly active, with a fondness for work and disinclination for reading and religion. Occasional spells of depression.

**Previous Psychotic Episode.**—Two years previously he had had a distinct phase of depression lasting two months. He did not receive hospital care.

**Present Illness.**—While at Camp Devens, a private in the infantry, he contracted influenza of moderate severity and duration. During convalescence he first developed apparently a short depressed phase, succeeded by increased activity and elation which necessitated his transfer to Boston. (Details on military cases were never satisfactorily obtained.)

**Mental Examination.**—He was an alert accessible, and loquacious young Jew of rather high empathic index; he talked very freely, with circumstantiality, frequent abrupt alterations of subject matter and manifestation of a crowd of ideas. No disturbance of orientation, memory, or thought content was demonstrated. He had a few fleeting phantasmagoric ideas which could scarcely be called either delusions or hallucinations; e. g., he said it "seemed as if all the girls with whom he had ever had sexual relations were in the palm of his hand." Gross conduct disorder was never observed; he was rather hyperactive, but easily controlled, and highly appreciative of efforts in his behalf, such as the prolonged baths, which he enjoyed immensely. Emotionally, there was a typical elation of mild degree.

Psychologic test rating: mental age, 16.

**Physical Examination.**—Well developed and well nourished; hyperactive reflexes, pseudoclonuses of ankles. Blood pressure: systolic 120, diastolic 70. Otherwise entirely negative.

*Laboratory Findings.*—Urine, blood cytology and serology, and spinal fluid examination complete, all negative.

*Diagnosis.*—Hypomania.

*CASE M.—Normality (?) + Influenza = Cyclothymic Psychosis, Manic Phase.*

*Family History.*—A boy, aged 17, was a student and was born in Massachusetts. His family history was negative in all respects, including his grandparents. There were four siblings, all living and well, and no miscarriages.

*Past History.*—This was carefully obtained in the outpatient department of this hospital when the patient was about 12. It was in all respects entirely negative. His birth, infancy and childhood were entirely normal. He talked at 8 months, walked at a year, attended kindergarten, grade, and high schools and did good work. He was studious but not seclusive and evidently had a rather superior personality. He was athletic and interested in tennis, swimming, etc.

From the age of 7 until the age of 13 he was troubled by stammering. He attended a class for speech impediment in the outpatient department of this hospital, and was very much improved after a year or so that he discontinued his attendance. Except for this his medical and mental past history is entirely negative.

*Present Illness.*—He was working hard at various school activities in a boys' academy, having many duties in addition to his curricular work. Besides a scholarship, he had earned some \$300 at outside activities, and had kept in excellent health until October 1. At that date he contracted influenza and was very ill, running a temperature of 105.5 F. for three days. He was able to be out by the sixth day, but a cough and much restlessness continued. He played tennis, went automobiling and took a short vacation, but continued to show a distinct hyperlogia, making extensive plans for the immediate and distant future. On the twelfth day this became very noticeable. He "talked rapidly from one subject to another," spoke of being nervous and wondered if he wouldn't go crazy. He was given morphin by a private physician, but without apparently much effect. His hyperactivity continued, and he was brought to this hospital at 1 a. m., October 13.

*Mental Examination.*—"The patient is rather tall, slender, attractive youth of 17 who is hyperkinetic, talkative, and elated. He confers various military ranks on the physicians in the hospital, elects the nurses to the Red Cross service, and announces large salaries for the attendants, etc."

"I am absolutely perfect. Have a cigaret? Here are two strings which they gave me for a test. Hello there, Major. We are all going to be in uniform before night. How old are you? I am 17 years and 9 months today and in 3 months I will receive a commission. . . . Girls? Yes, girls by the thousand. Girls from Wellesley, girls from Dartmouth . . . no, there are no girls from Dartmouth . . . girls from Smith, girls, girls. We'll put this thing across, and have all those beds put in. Can you see it? Will you help it? Never mind, not necessary." (Whistles!)

The patient showed hyperlogia and hyperactivity as were indicated from the day of admission until discharge on the tenth day. He amused himself and his audience greatly, performing antics unnumbered, and of infinite variety, particularly when in the prolonged baths, and particularly when observed. *Emotional tone* was that of elation, exaltation, euphoria. His *thought processes* showed typical flight of ideas, with distractibility, play on words, etc. His *associations* were easily followed, however, for the most part. *Delusions* were

fleeting, and of the expansive and grandiose type, never paranoid, somatic or referred. There were no *hallucinations*. The *conduct* was as described above; hyperkinesis over a wide range, but usually readily controlled. He was obedient and not destructive.

*Diagnosis*.—Cyclothymic psychosis, manic phase. Committed.

CASE N.—*Normality (?) + Influenza = Cyclothymic Psychosis, depressed phase.*

*Family History*.—This patient was a man, aged 24, single, a chauffeur by occupation and was born in Boston. His grandparents were all born in Ireland, and died there at well advanced ages. His father was born in Ireland, a citizen of United States, a freight-handler, aged 51, without abnormalities of mental life. His mother was born in Ireland, aged 48, a charwoman, quite healthy. Possibly both mother and father are at least mildly alcoholic. Siblings: of parents, no social or mental pathology. Of patient, 8, 3 dying in infancy of enteric diseases, the others living and well. No history of mental pathology anywhere in the family. Excessive alcoholism also denied.

*Past History*.—The patient was born in Boston in 1894, and according to the history furnished by mother was evidently a normal infant and child. He left school at the seventh grade to work, was 14 and was considered a good student; had not been held back.

His *economic* history is rather varied, but not important. For some years he had worked as chauffeur, earning from \$20 to \$25 weekly.

*Personality*.—"He has a happy, sociable disposition. Has been very good to his mother, giving her most of his wages. Is fond of motion pictures, Chinatown suppers, and social parties. Is much liked, and is not suspicious." Catholic. Habits: Occasionally drank beer; "never seen or suspected of being drunk." Medical: Scarlet fever at 4, very severe and protracted. Diphtheria at 7. Since then never sick, until present illness.

*Present Illness*.—Ten weeks prior to admission he is said to have had influenza. "He was very ill. Temperature could not be taken because his teeth chattered so. He was in bed 7 or 8 days. He had night sweats (thereafter) for about a week."

He returned to work after two weeks, "but looked pale and weak but felt pretty well." He remained at work a week and "had a relapse." "He had a chill, sweats," and probably fever. He was confined this time for nine days, but was not in bed throughout this time.

Thereafter he felt quite incapable of returning to work. He "thought that he was dying," that "some one had given him the disease a second time," that "he couldn't seem to take hold and do his work." This continued for two weeks.

Two days before admission he called to his mother, asking what it was that was in bed with him, that it had frightened him. He was not suicidal, however, nor did he show conduct disorder nor react more definitely to hallucinations.

*Mental Examination* (abridged).—He wore constantly a pained, doleful, anxious expression. He was accessible, and tried to cooperate, but was not very successful because of his aboulia, retardation, and a suggestion of impoverished, intellectual processes. *Orientation* precise in all spheres. *Memory* general, rather faulty, perhaps due in part to abstraction. No *hallucinations*. He had an imperfectly formulated *delusion* that he had recently contracted venereal disease. No others. There was partial *insight*. . . . "Sometimes I don't seem right." His *thought processes* showed a conspicuous retardation, with

rather weak associations and an attention difficult to secure or retain. His *motor status* was distinctly that of hypokinesia and abulia. He sat about the wards all day, with head hung, and without demonstrating interest in anything. When obliged to move he did so with slow, irresolute movements, and with an air of lugubrious torpor. His *emotional tone* was incontrovertibly that of depression. He was "blue," "lonely," "worried," "down-hearted." He was never seen to weep, or to smile; he commented once that "some days things seem dark, and other days brighter."

*Physical Examination.*—This was entirely negative except for exaggerated reflexes and a tremor of the hands.

*Laboratory Examinations.*—These were negative, including spinal fluid and blood Wassermann tests.

*Results.*—His condition remained unchanged. He was removed against advice on the ninth day.

*Diagnosis.*—Depressed phase of cyclothymic psychosis (manic depressive, depressed).

#### GROUP 10.—*Psychoneuroses*

There is a book (4 vo) written in 1890, and now little read, entitled "De l'hystérie consécutive à la grippe" (Le Joubioux<sup>43</sup>). The phenomenon referred to is striking and frequent enough to have attracted attention after each of the later epidemics of influenza. Grasset, Krannhals, van Deventer, Worms, Josserand, Huchard and many others have contributed reports and discussions, the consensus of which is well put by Leichtenstern.<sup>16</sup> "Hysteria and neurasthenia not infrequently arise from influenza or are enormously exaggerated by it. All forms of hysteria have been observed. . . ."

He then asks the important question, "Is a neuropathic predisposition the basis of hysteria and neurasthenia when following influenza?" and answers "probably . . . in the affirmative, in most cases. On the other hand . . . severe postinfluenzal hysteria has frequently been observed . . . (where) no hereditary or acquired neuropathic tendency could be found." In this he simply speaks for a number of observers, some of which he quotes.

To circumvent the question, I present herewith two cases, one having a definitely manifested predisposition to psychoneurotic episodes, the other quite without any such tendency.

*CASE O.—Psychoneurotic Tendency* (manifested by a previous "hysterical attack") + *Influenza* = *Psychasthenia*.

*Family History.*—The patient was a woman, aged 34, born in Cambridge. Her paternal grandparents were Irish, both lived to be over 90; always healthy and temperate. Her maternal grandparents were Irish and English; both died "of old age" in the eighties; also healthy and temperate. Her father was born in Ireland, and came to United States when 12 years old. He died at 71 of

43. Le Joubioux: Thèse de Paris, 1890.

"cancer of the intestines." He was a railroad night-watchman, temperate and healthy. Her mother was born in Ireland and is living and well, aged 73. Paternal siblings, 9; maternal siblings, 8; all are living and well. There had been one miscarriage, at 6 months, during typhoid fever.

*Past History.*—The patient was born in Cambridge, Oct. 29, 1884. It was a normal delivery, a healthy baby, and she passed through a normal childhood. Puberty occurred at 14 or 15 without disturbance or illness.

*Education:* She entered the kindergarten at 3. Went through grade schools and finished the second year of high school at age of 15. Considered "very bright" and had "double promotion" twice.

*Economic history:* On leaving school worked two years as a night telephone operator. Mother objected, and so she remained at home for a year or more. From 21 to 30 she was employed as a saleslady by one firm, at \$10 a week. Has not worked steadily since (*vide infra*).

Not married. No court record. Habits normal. Religion: Catholic, devoutly held. Medical history negative except for the surgical removal of a small cyst and the appendix in July, 1917.

Personality described as "sympathetic, very sensitive, but not suspicious or jealous. Unselfish, very sociable, never irritable or critical. Fond of music and singing. (The mother, who gave the account, adds that "for a year I have not let her play because it seems to make her nervous.") It is thought that she was influenced rather with difficulty.

*Previous Episodes.*—The patient is well known to the outpatient department of this hospital. Four years previously she suddenly became "hysterical" at the sight of a brother dying with tuberculosis. She failed to recognize him [*sic*], cried "uncontrollably," and remained depressed and lugubrious. Her own account is identical, in that she explains that she was so "shocked" by her brother's appearance and so appalled at her helplessness in the situation that she had "an hysterical collapse" and was led from the room "screaming and crying." She was not able to work thereafter with any degree of proficiency. Although better at times, and particularly when under the surveillance of the social service department, she was nevertheless prone to "attacks" of profound despondency and weeping, once a month or less frequently. The social service worker finally established her at knitting on a commercial basis in her own home and she was diligent and efficient thus employed, knitting over fifty sweaters in the past year, and coincidentally feeling better than previously. She was consistently and persistently followed by the outpatient department and a voluminous literature has accumulated in her folder. The diagnosis there made was psychasthenia. She was considered much improved and had not been seen for nearly a year by the physicians.

*Present Illness.*—On October 10 she contracted influenza. "She had a temperature of 102 and 103 F. for the first few days. The fever did not pass for ten days; she was in bed about three weeks. For about two days, at the beginning, she was out of her head." Thereafter she seemed much the same for three weeks. On November 21 she complained of severe headache "and mumbled things to herself. She seemed much distressed and afraid of going crazy." She was brought on the following day to this hospital.

*Mental Examination.*—A young woman of rather large frame, well nourished, attentive, accessible, and cooperative, but very much self-centered. Consciousness entirely clear at all times, speech normal, orientation precise, memory free from defect. She gave a rather more detailed account of her past than that

furnished by the outside informant, but the main facts coincided throughout. She denied any stigmata of masked epilepsy (enuresis, somnambulism, etc.). Obsessive, obscene thoughts began to trouble her shortly after the attack of four years ago. She said they did not seem to originate from any particular cause of experience and they persisted for a year and a half. After the consultations in the outpatient department this improved and she was comparatively free from these thoughts.

Her account of her present illness is as follows: For some time she has been fairly comfortable mentally and physically and busily engaged in knitting sweaters at home, proceeds from the sale of which afforded her some little income. After a prolonged attack of influenza she began again to be troubled by obscene thoughts "much more than ever before." She told no one about them for two weeks, but they became so distressing that she could endure it no longer and following an expression of her feelings she was brought to this hospital. These thoughts are obscene and sexual suggestions and phantasies which "possess her whenever she is approached by any person." They come in spite of her dislike for them and her avowed efforts to keep them from her mind. They seem to have no particular reference to certain types of persons, they were not accompanied by hallucinations. "Something seems to grip my brain." After the thought comes the regret and chagrin that she should have been guilty of such thoughts. They seem to come in distinct showers; thus "today had none until about 3 p. m., when on roof garden" and "frequently has them after retiring at night." Later the obscenities became related to religious matters, but she was unable to explain this adequately.

In addition she had some symptoms of the "*folie de doute*." Thus for example she said "Sometimes I seemed compelled to repeat sentences, things I hear. If some one says breakfast is ready I feel as if I must keep repeating to myself, 'breakfast is ready, breakfast is ready.'"

She was precisely oriented, showed no memory defect, no hallucinations or illusions, no delusions, good insight and judgment, a rather variable emotional tone swinging from depression when introspective to pleasant, cheerfulness when distracted, normal thought processes and motor status. She was distinctly suggestible and was considerably improved by unsystematic suggestive therapeutics.

*Physical Examination.*—This proved to be essentially normal in all respects. Reflexes were slightly hyperactive; blood pressure was systolic 140, diastolic 80.

*Laboratory Findings.*—These were negative as to urine, blood serum, spinal fluid and vaginal smear.

The patient was discharged considerably improved after two weeks, with the diagnosis of psychasthenia.

CASE P.—*Normality + Influenza = Hysteria.*

*Family History.*—A girl, aged 15, was a student, and was born in Massachusetts. Her father was living and well, aged 52, not alcoholic. Her mother, aged 49, was always very healthy except for a gradual increasing deafness for the past fifteen years. "She has always been rather nervous and high strung and recently more irritable than usual." The patient stated that the entire family were "rather nervous and easily excited," but there was no history of nervous, mental, or epileptic disease in the family. One sister, aged 18, is healthy. Two siblings died in infancy of unknown causes.

*Past History.*—The patient was born in a suburb of Boston in 1903. She was rather weak and "sickly" until the age of 5, but since that age had enjoyed

unusually good health. Aside from the usual diseases of childhood and an occasional "cold" she had never been ill. There was no history of convulsions or other stigmata of epilepsy.

**Educational:** She started going to school at the age of 6 and was at the time of examination in the third year of high school. School work was always done very well and with genuine interest. Although she found her studies easy she was rather too assiduous and studied late into the night.

**Personality:** She seems to have been always a cheerful, happy individual with many friends and with one particular chum with whom she had associated closely for three years. Although her interests seemed to be confined chiefly to her school work she was definitely social, enjoying a crowd and disliking very much to be alone.

**Present Illness.**—The patient herself gives a very good account of her present illness, and it is here reproduced with minor modifications. An attack of influenza began November 2. She was in bed for about a week. She had a temperature as high as 103 F. There was apparently a good recovery after this, but about two weeks ago, since she started back to school, she noticed that she tired very easily, and felt she was overworking. She admitted that she had been studying more than usual in order to make up for the time she had missed.

The day before coming to the hospital she felt particularly tired. She had been studying late the night before, preparing for an examination, and felt so tired the next morning that she did not eat any breakfast, and also missed her lunch while at school. That afternoon she went to her friend's house, and while there became even more worn out. She said she did nothing out of the ordinary that afternoon, but after she had been there awhile she became rather silly. She felt that she was rather excited, and would laugh without provocation, and would say foolish things. Soon, however, she quieted down, and while sitting and talking realized suddenly that her left leg felt as if it had gone to sleep. She could not feel anything at all—felt as if it were gone. Tried to stand up but it collapsed under her, and she saved herself from falling by grasping a chair. This frightened her and she thought that perhaps she was going to be paralyzed. Then the other leg began to feel the same way, but not so markedly. The doctor was called soon after this, and he said it was nothing but nerves, and that she would be all right. This comforted her and although she felt too weak to go to her home she spent a restful night at the house of her friend. The next morning she was feeling entirely all right except that her legs were sore, and she had a rather severe pain in her back. She endeavored to get dressed so that she could go to her own home, but the pain in her back and legs would increase on any attempt of movement. That afternoon she thinks she must have become delirious because there are periods concerning which she can remember nothing. She would come out of these confused periods, and have no complaint except the soreness in her back, but soon she would drift away into unconsciousness. Once or twice while she was unconscious she felt that she was blind. She could see nothing, but would talk to the people around her and ask them why the room was so dark. They told her afterward that the light was on and that they could see perfectly clearly. These periods of blindness lasted only a few minutes. She knows nothing about what she said or did during the periods when delirious, although since she has been told about it by her family. She was afraid this time that they were going to take her away, send her to some hospital, and she would not be able to see her friends. She thinks she probably talked about this during the delirious periods.

She still was rather confused and uncertain when she came to this hospital, but the following morning felt entirely normal and has been that way ever since. She tells also of "an occasional lump in her throat, causing her to nearly choke." When asked about her girl friend, she admitted that she cared for her very much, but denied anything of a homosexual nature in their relations. Nothing of this kind could be elicited in any way. She admitted she had done a great deal to further the match between her friend and her cousin who is in France. She even wrote letters to her friend and signed them with the name of the man who is in France, but said she told her friend about it a month or so afterward. There had been no deception for at least three months on her part. She did not know whether the girl had been deceiving her or not.

*Mental Examination.*—Orientation precise, memory absolutely unimpaired except for certain recent events as detailed above. No delusions, no hallucinations, good insight. Thought processes show excellent attention, quick and coherent associations, normal train of thought, speech free from fault. Emotionally cheerful and pleasant. Conduct above reproach.

*Physical Examination.*—Entirely negative in every respect. The same is true of the laboratory examinations which included spinal fluid, blood serum, urine and vaginal smear.

*Psychologic Examination.*—The patient graded regularly at the mental age of 18 plus. Her performance on the construction puzzles was fairly good and on the memory tests good. In the suggestibility tests she accepted ten of ten suggestions. Patient cooperated well.

She showed no further evidences of mental disease during her stay here. She gave the impression of being "high strung" and "of an hysterical type" to various physicians who examined her. She was discharged improved after five days, with a diagnosis of *hysteria*.

#### GROUP 11.—*Psychopathoses*. Psychopathies, and the unclassified

It was Southard's intention that in this group there be included not only those cases of dubious mental pathology who are variously classified as "constitutionally inferior," "psychopathic personality," "defective delinquent," etc., but also those occasional instances of psychotic forms entirely aberrant from any recognized type or group. These cases in which there is no possibility of agreement on a comprehensive and justifiable diagnosis of any kind are relatively infrequent, but they are common enough to be well known in private practice as well as in institutional work.

The following instance is perhaps not the best example, but it serves in a measure because of the strong suggestion of psychopathy and because of the precipitation of symptoms by influenza, although the influence of the latter is equivocal. I am aware of the similarity in some respects of this case to those described by Grasset,<sup>44</sup> Krannhals, and in the German army reports, as mentioned by Leichtenstern<sup>10</sup> and epitomized as "the so-called hystero-epileptic [sic] attacks, occasionally

44. Grasset: *Leçons sur la grippe*, Paris, Masson et Cie; Montpellier, Coulet, 1890.

associated with fits of crying." Nevertheless, I agree with the intimation therein put forth that the demonstration of the hysterical nature of those phenomena (even if this case be proved to be similar) is not wholly convincing.

CASE Q.—*Psychopathic Basis (+?) + Influenza = Undiagnosed Psychosis.*

*Family History.*—The patient was a married woman, aged 35, whose occupation was housework; she was born in Nova Scotia. Her grandparents' history was negative except that "patient states that she heard her father say that his mother lost her mind after her son had been killed." Both parents were living. Her father was 60, mother 66, father well, mother crippled by "rheumatism." Siblings, four living and well, three died in childhood (scarlatina, drowning, and "unknown"). No history of mental disease in family.

*Past History.*—She was born in Nova Scotia in 1884; full term, normal delivery. She passed a normal childhood, except for somewhat inferior physical status. "She was always the delicate one of the family." Had measles and scarlatina; later "chronic bronchitis." Was "always nervous."

*Educational:* There is some discrepancy in the accounts of her schooling, but at any rate she was a rather superior child and is said to have been in the eighth grade at the age of 13. She left high school at the end of the first year to get married. Her school work seems to have been very good, and she was not held back.

*Economic:* She worked for about a year just prior to marriage, at a Boston department store. Other than this she never did anything but housework.

*Marital:* Married at 16 to a private detective who never properly supported her and who eventually deserted her when her youngest child was 5 weeks old. She later obtained a divorce. He alleged immorality on her part (while married to him), which she denies. There were three children, all living and well, aged 16, 14 and 11. In addition there were several self-induced miscarriages.

Subsequent to her separation from her husband she was sexually intimate with two men, whose names she gives. These indiscretions had an important relation to her present illness.

*Previous Nervous Trouble:* Five years previously she was in the outpatient department, because of insomnia, worry over children who were in custody of divorced husband at that time, paresthesias of legs, tingling in hands, supposed loss of memory, inadequacy, etc. Given a few hydropathic treatments; improved.

*Character:* Not seclusive. Not much adequate information secured because of prejudice of informants (including patient).

*Present Illness.*—Influenza began October 9. She was ill only two days. "Following this, however, she was unable to sleep and she could not think right. She has great difficulty in recalling what she did the next few days. Then she commenced worrying over her recent sex indiscretions. To add to her troubles a young niece died of influenza on the 12th."

She was brought to this hospital on the 18th by her father. The admission note reads as follows:

"In admission office the patient at first refused to answer questions. Then said there was no reason for her coming here. Is afraid that she is going to be killed here. Does not show any marked emotion over it, however." Later in the ward, "she is pleasant and accessible, and does not appear apprehensive

or depressed. No hallucinations or definite delusions. Says she had influenza following which she became depressed and retarded; now complains of feeling weak."

*Mental Examination.*—*Orientation* precise in all spheres. *Memory* intact except for events just preceding admission, which are apparently quite hazy. *Ideation* characterized by self-accusatory ideas; for example, she had disgraced her children by her sexual indiscretions; the nurses knew of her miscarriages from her chart and would look down on her; father and mother would find out about her, or be informed of what she had done. There was no *insight*, nor on the other hand any definite delusions. *Hallucinations* absent. Thought processes showed no irrelevancy, incoherency or retardation. *Emotional Tone:* Except for periods as described below, her emotional tone seemed to be quite normal; she was cheerful and pleasant and responded to interrogation freely and cooperatively. The attacks described were accompanied by wailing and followed by singing; it was impossible to determine what or how she felt emotionally.

*Motor Status.*—The characteristic feature in the case was the frequent outbursts of wailing, shrieking and weeping, accompanied by a clonic stiffening of the muscles of her entire body, and a flushing of the face. These attacks were of perhaps twenty minutes' duration; they were sometimes induced by interrogation on the matter of her indiscretions, but at other times she answered the same questions with no disturbance whatever. The manifestations differed slightly at different times, but the essential things were complete inaccessibility because of a lugubrious demonstration which was in some cases followed by equally inexplicable elation. At one time during an attack, when she was stiffened out tensely, a pinprick test was attempted and she was found to be anesthetic.

She herself explained these attacks on the basis that she "felt so degraded" to think she would be suspected of immorality. Yet this she freely admitted, and at the same time insisted that she was sexually frigid.

Still another type of conduct disorder appears in the following interrogation, which also gives a good idea of her type of response: (One night you did a great deal of screaming, and pounding on the door.) "No, it was morning."

(What time?) "It was after breakfast. I had spent an awfully restless night and I went in and asked Miss P. if I could lie down. She just took me and threw me into the cells there, and I got into a kind of frenzy."

(We have no cells here.) "I thought it was locked. She slammed the door so hard that I thought she locked it."

(Even if she had locked it?) "I got awfully frightened. I have a home and three children and I want to go home to them."

(Are you afraid to be left alone with yourself?) "No, I am perfectly safe alone."

(I don't see why you should be so frightened?) "Just a terror came over me."

*Physical Examination.*—General hyperalgesia. Positive Romberg. Reflexes normal, except knee jerks which, although equal, were diminished. Blood pressure, systolic 118, diastolic 62.

*Laboratory Findings.*—Urine, blood serum Wassermann test, spinal fluid (including Wassermann test), vaginal smear and complement test for tuberculosis all negative.

*Psychometric Test.*—The patient graded regularly at a mental age of 18. In the supplementary tests her performance in the construction puzzles was

good. The memory tests were also well done. She showed fairly good comprehension and judgment in the apperception picture puzzle. She *accepted* two out of ten *suggestions* [sic]. She cooperated well.

She showed some improvement, but was committed.

The diagnoses made here ran the gamut of psychiatric possibilities, including dementia praecox, psychopathic personality, and psychoneurosis. Against the first named is preeminently the absence of schizophrenia. Against psychoneurosis there is less definite proof, but it might be pointed out that her conduct was much disturbed, that insight was completely lacking, that it was not proved that she was hypersuggestible or bore stigmata of hysteria at all, and finally that she was committed. The possibility of cyclothymic psychosis may be suggested, but the absence of any constant depression or elation, and the absence of retardation at all times, makes this rather unlikely.

#### SUMMARY

The effect of influenza on the brain may now be considered analytically by summarizing the cases collected and detailed in the foregoing.

There are obviously two methods of approach: One may consider in summary merely the end results, which being the matter of primary pragmatic value is usually considered first. Complementary to it, however, is the study of the component essentials, the basis and contributing factors which together bring about the final product, a study of the material on which the influenza acted to produce specific results.

Considering the first method, one may tabulate the paradigms to show the end results thus (in Southard's order):

*Apparent Normality + Influenza = General Paresis (Neurosyphilis).*

*Morosis + Influenza = Imbecility (Hypophrenia).*

*Epilepsy + Influenza = Alteration in Frequency and Type of Epilepsy.*

*Alcohol(ism) + Influenza = Delirium Tremens (Etc.) (Alcoholic Psychoses).*

*Normality + Influenza = Cerebral Hemorrhage (and Psychosis).*

*Normality + Influenza = Delirium (simple, errant, schizophrenic, etc.).*

*Senescence + Influenza = Senile Psychosis.*

*Normality  
or  
Predisposition* } + *Influenza = Dementia Praecox (Schizophrenia).*

*Normality  
or  
Predisposition* } + *Influenza = Manic Depressive (Cyclothymic) Psychosis.*

*Normality  
or  
Predisposition* } + *Influenza = Psychoneurosis (Hysteria, Psychasthenia, etc.).*

*Psychopathy + Influenza = Psychosis (Atypical).*

From this presentation one is justified in the conclusion that any form of mental disease may follow influenza. But this is to declare a chronologic relationship only, and to imply or assume an etiologic connection that only a study of the component elements of the equation can justify or elaborate.

In a sense, we should be justified in urging in place of the phrase "influenzal psychoses," which literally do not exist, the more cumbersome but more correct expression, "influenzal neurotoxic effect (or product)." Surely the most striking and noteworthy fact is the variety of psychic (and encephalopathic) lesions. This fact alone is particularly and specifically intolerant of a doctrine of specificity. This we may say without reference to the modifying or even determining factors contributive in each case or group of cases, simply because of the great inclusiveness of the list. A disease—an incident—which is capable of calling forth psychiatric pictures as widely different as general paresis and hysteria (I temporarily waive the point that in neither case cited as illustrating these phenomena was there any previous indication of mental trouble), and which does not fail to leave its conspicuous impression on every intervening group of mental disease in a representative list, can surely not longer be accused of possessing psychic specificity.

Reversing the point of emphasis, now, and considering minutely the left-hand side of the equation, the components of the sum, we may tabulate the cases in three groups. Primarily, there are those arising on a basis of undisputed psychic normality. In these the left-hand side of the equation reads throughout:

$$\text{Normality} + \text{Influenza} =$$

These are distinctly psychoses of *creation*, as opposed to psychoses of precipitation, revelation or alteration. I have suggested these terms as descriptive of the process involved merely as useful figures, and without presuming to indicate what psychopathologic process actually takes place. It is established that this is what *seems* to take place, and these then are the processes that seem to be represented. The first one, *creation*, is represented by the following paradigms:

$$\text{Normality} + \text{Influenza} = \text{Delirium.}$$

*Cerebral Hemorrhage with Psychosis.*

*Senile Psychosis (?)*

*Schizophrenia (Dementia Praecox).*

*Cyclothymia (Manic-Depressive).*

*Hysteria (Psychoneurosis).*

The next group of equations are those in which the influenza acts on an avowedly predisposed soil, resulting in the production of a

psychosis of a specific form, exactly analagous to those mentioned in which no evidence of predisposition was shown to exist. These cases are based on equations the left side of which is uniformly

$$\text{Predisposition} + \text{Influenza} =$$

They represent a process to which scores of verbs have been applied. The French and German terms are scarcely less numerous than the English, and "provoked," "incited," "instigated," "produced," "called forth," and "excited" are only a few of these. The inference is that the nature of the process is frankly not understood. If I prefer to use the term "precipitated," it is not with the idea that it expresses any better conception of the nature of what takes place. Elsewhere there will appear shortly a more presumptuous attempt to liken the process to that of catalysis in chemistry, but I do not urge this here. This group, representing the process, we will say, of *precipitation*, is represented by the following paradigms in the above cases:

$$\text{Predisposition} + \text{Influenza} = \text{Delirium Tremens (etc.).}$$

*Schizophrenia.*

*Cyclothymia.*

*Psychoneurosis.*

Finally, there is a smaller group representing the power of influenza to alter the nature or degree of a neuropathologic or psychopathologic lesion already extant. These cases may masquerade under guises suggestive of a process primarily of revelation, such as the revelation of a heart lesion by an attack of pneumonia. Thus the appearance of general paresis immediately after influenza might simulate this, but actually it probably amounts to but an augmentation or hastening of a process no doubt already established, and possibly capable of detection were all the refinements of diagnosis possible of application in an unsuspected subject. This group, representing the power of influenza in the *alteration* of pathologic brain processes, is represented here by

$$\text{Morosis} + \text{Influenza} = \text{Imbecility.}$$

$$\text{Epilepsy} + \text{Influenza} = \text{Alterations in frequency and type.}$$

$$\text{Psychopathy} + \text{Influenza} = \text{Psychosis.}$$

$$\text{Apparent Normality (Latent Neurosyphilis)} + \text{Influenza} = \text{General Paresis.}$$

$$(\text{Mild Neurosyphilis} + \text{Influenza} = \text{Advanced Neurosyphilis}).$$

Influenza thus acts apparently in a nonspecific manner to do specific things; to create psychoses, to precipitate them in predisposed persons, and to augment or alter them where already existent. So powerful and so versatile a neurotoxin is certainly not possessed by any other acute infection. It is conceivable, although scarcely so, that were there

some other potent infection as ubiquitous as influenza, its neuropsychiatric effects might be as numerous and as various, but certainly evidence does not favor this. It is not true of the sequelae of the great plagues of typhoid and bubonic plague that once raged; lobar pneumonia is certainly common enough and widespread enough to have resulted by this time in a similar host of nervous sequelae were it akin to influenza in neurotoxic effect. Quantitative specificity, in point of high potentiality in neurotoxic effect, is apparently fairly claimed by influenza.

Finally, the writer would add a word in defense of the equation paradigms as used in this article. I am well aware, of course, that it is impossible to accurately and wholly express any biologic process in chemical or mathematical formulas. They have been adopted here, not for expressing the whole truth, even if anyone grant that to be attainable, but the essential truth as concerned with influenza, the human basis on which it works and the psychosis which succeeds. There are doubtless many elements entering into the formulas expressing influenzal action on human brain which are not represented in the paradigms of this article, elements which while absolutely indispensable to the complete configuration of the process, are in no way essential to the basic principles represented by the simpler and wholly pragmatic cryptograms.

#### CONCLUSIONS

1. This paper aims to illustrate the forms of psychoses associated with influenza by the presentation of representative cases.

2. The history of the study of the association of mental disorder and influenza is traced roughly from its inception in 1385 to its frank recognition in 1790 and its elaboration since then.

3. Southard's Eleven-Group nosology is commended by its mechanical convenience and its neat inclusiveness, and serves admirably to present cases illustrative of findings as follows:

4. Active neurosyphilis (Southard's Group 1) may be precipitated by influenza (and cases of sluggish course accelerated). (Case A.)

5. Hypophrenia (Group 2) may be augmented in degree, and a case of the apparent process "Morosis + influenza = Imbecility" is cited, but no evidence was obtained for the production of total loss of intellect, the acute dementia of the idiocy type, or Kraepelin's misnamed "infectious idiocy." (Case B.)

6. Epilepsy (Group 3) may be altered quantitatively and qualitatively, that is, in the frequency and in the form of attacks, but there were no instances of its initiation by influenza in our series. (Cases C and D.)

7. Delirium tremens and other forms of alcoholic psychoses (Group 4) were quite frequently induced by the added toxemia of influenza, but probably in no greater frequency than would obtain in a similarly large number of any acute infectious disease. (Case E.)

8. Of the encephalopathic psychoses (Group 5) the occurrence of Leichtenstern's influenzal "hemorrhagic encephalitis" with a peculiar psychosis was demonstrated clinically and by necropsy. (Case F.)

9. Delirium (Group 6) remains the most polychromatic and versatile of mental disease pictures; its association with influenza is notoriously frequent, and its manifestations bewilderingly multiform. It stands as the type illustration of the paradigm, "*Unknown factor + influenza = psychosis.*" (Case G.)

10. Of psychoses associated with senility and the presenium (Group 7) one rather equivocal case is presented as having been initiated by influenza without previous indications. (Case H.)

11. Schizophrenia (Group 8), cyclothymic psychosis (Group 9) and psychoneurosis (Group 10) occur following influenza with and without predisposition or previous manifestations. Instances of all are given in detail (Cases I, J, K, L, M, N, O and P.)

12. Undiagnosed psychoses and psychopathias (Group 11) form rather too vague a group to be considered categorically, but a representative case is given in which influenza incited a psychotic episode in a (?) psychopath. (Case Q.)

13. The cases presented may be summarized by paradigms, exemplifying the psychiatric effects of influenza, viz.:

(a) In the process of *Creation*:

*Normality + Influenza = Delirium (simple, errant, schizophrenic).*

*Apoplexy, Atypical Psychosis.*

*Senile Psychosis (?)*

*Schizophrenia.*

*Cyclothymia.*

*Hysteria.*

(b) In the process of *Precipitation*:

*Predisposition + Influenza = Delirium Tremens (etc.).*

*Schizophrenia.*

*Cyclothymia.*

*Psychoneurosis.*

(c) In the process of *Alteration*:

*Morosis + Influenza = Imbecility.*

*Epilepsy + Influenza = Alterations in frequency and type.*

*Psychopathy + Influenza = Psychosis.*

*Apparent Normality (latent neurosyphilis) + Influenza = General Paresis.*

*(Mild Neurosyphilis + Influenza = Advanced Neurosyphilis).\**

14. Influenza apparently acts on the brain in three ways: to create psychoses, to precipitate psychoses in predisposed subjects, and to augment or alter their form where already existent.

15. Thus we cannot from the present data regard influenza as capable of *qualitative* psychic specificity.

16. The *quantitative* specificity, however, of the influenza neurotoxin is confirmed by its remarkable potency and versatility; the large number and wide variety of psychic and encephalopathic lesions produced being one of the most striking neuropsychiatric features of the epidemic.

17. The question of predisposition is simultaneously answered, there being evidence to show that psychoses sometimes occur directly after influenza with no forerunning symptoms or signs, and sometimes occur then only after months or years of less pronounced manifestations.

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\* Mentioned but not detailed in this article.

## SENSORY CHANGES IN PERIPHERAL NERVE INJURIES

LEIZER E. GRIMBERG, M.D.

Assistant, Neurological Institute

NEW YORK

The present article is based on a study of peripheral nerve injuries for a period of one year, during a part of which time I was stationed in France, and the balance of the time in this country. A large number of the patients were examined soon after the injury; many of them a year or two after the operation and others at various periods after receipt of the wound.

### 1. THE SENSORY DISTURBANCES IN PERIPHERAL NERVE INJURIES

The sensory disturbances in peripheral nerve injuries do not always go hand in hand with severity of the injury. This probably depends on the anomalies often present in sensory distribution, anastomosis (especially between median and ulnar) and the location of nerves supplying adjacent areas. I frequently encountered complete sections—found so at operation—showing slight sensory disturbances; and often incomplete or partial injuries of a nerve with extensive sensory disturbances. The first applied especially to the musculospiral nerve, the second to the ulnar. The musculospiral nerve is preeminently motor. Occasionally only a small area of anesthesia will be found around or over the first dorsal interosseous space. At other times it extends to the forearm, encroaching on the musculocutaneous area. A slight injury of the ulnar nerve is liable to give extensive sensory disturbances, frequently passing beyond the typical ulnar area. Rarely, complete section causes anesthesia just over the dorsal surface of the fifth finger. A slight injury of the external popliteal nerve may give extensive areas of sensory disturbances, while complete section of the sciatic nerve may produce less extensive changes.

I have selected for the first part of this article ten cases which gave the syndrome of complete interruption, subsequently corroborated by operation. This syndrome was the result either of complete nerve section or interruption by scar tissue, fusiform growth, etc., so that nerve fibers, if present, were degenerated. Therefore, in this article "syndrome of interruption" means that no healthy and acting nerve continuity exists.

The following illustrations were used to represent the various sensory changes:

CASE 1.—L. P., received a gunshot wound in the outer side of the thigh; the upper third. Findings: The sciatic nerve was found with a large bulbous enlargement 5 cm. below the gluteal fold. Faradic stimulation below the bulb did not elicit response. Operation was performed five months after injury. Two months after operation there was no motor improvement. Sensory changes were as indicated in Figure 1.

CASE 2.—H. L. received a gunshot wound, antero-internal aspect; the lower third of the left thigh. Findings six months after injury: The sciatic nerve was bound down by dense scar tissue. The internal popliteal portion showed a bulb for a distance of about one-half inch. Sensory Figure 2 was obtained several days before operation. The existence of a small area of hyperalgesia made the diagnosis of a complete section doubtful.

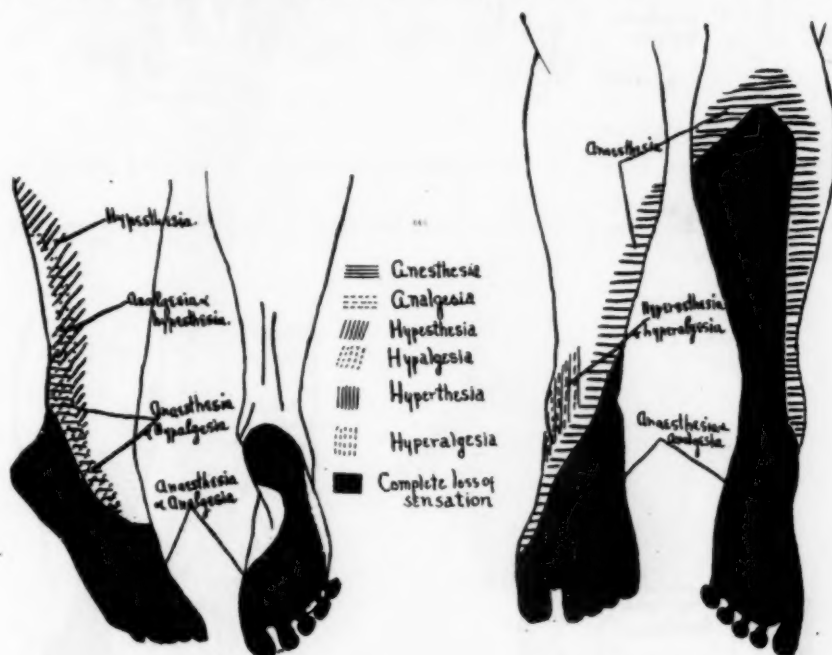


Figure 1

Figure 2

Fig. 1 (Case 1).—L. P., peripheral nerve injuries following gunshot wound of the thigh.

Fig. 2 (Case 2).—H. L., peripheral nerve injuries after gunshot wound of the thigh.

CASE 3.—F. J. received a gunshot wound, antero-external surface; the middle third of the left arm. Findings at operation five months later: The spiral nerve was separated for a distance of  $1\frac{1}{4}$  inches. Sensory Figure 3 was made several days before operation.

CASE 4.—C. C. suffered a wound of the right axilla due to a hand-grenade explosion. Findings: The median nerve was separated for a distance of one-half inch. The ulnar nerve could not be freed from scar tissue. Examination was made ninety-six days after nerve suture. Regeneration of the median nerve had begun, but there was no improvement in the ulnar. Clinically we had the syndrome of complete interruption of the ulnar nerve (Fig. 4).

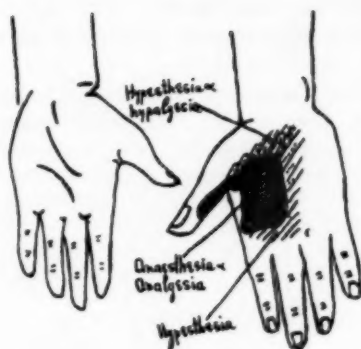


Figure 3

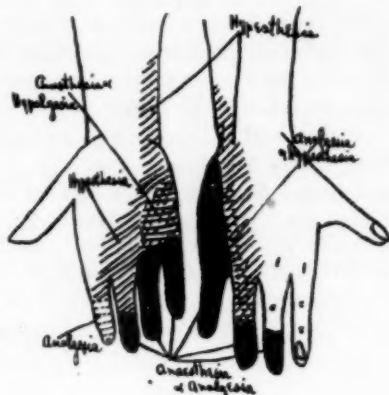


Figure 4

Fig. 3 (Case 3).—F. J., nerve injuries resulting from gunshot wound of the arm.

Fig. 4 (Case 4).—C. C., nerve injuries from a hand-grenade wound of the right axilla.

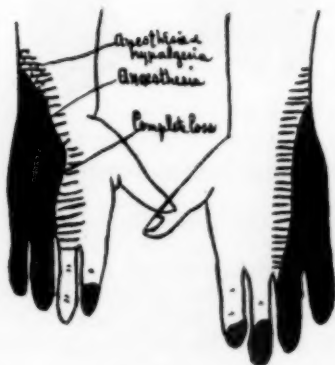


Figure 5

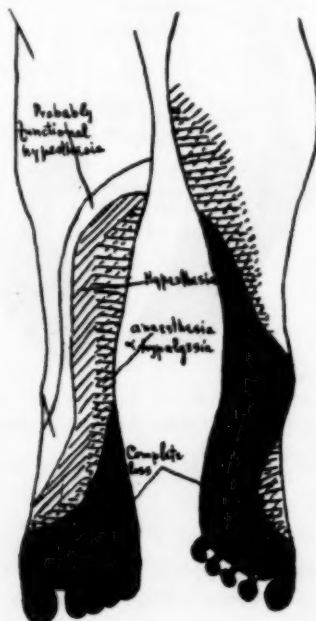


Figure 6

Fig. 5 (Case 5).—L. J., nerve injuries following a gunshot wound of the arm.

Fig. 6 (Case 6).—Z. A., nerve injuries after high-explosive wound of the thigh.

CASE 5.—L. J. suffered a gunshot wound, anterior aspect; lower part of the arm. Findings at operation ten months later: The ulnar and median nerves were divided. The ulnar was separated for a distance of three-quarters inch, and the median for  $2\frac{1}{2}$  inches. Figure 5 was made one month before operation. The minor sensory changes in the median area, and extensive sensory disturbances in the ulnar region should be noted.

CASE 6.—Z. A. suffered a high-explosive wound of the lower third of the left thigh. Findings at operation six months after injury: The external and the internal popliteal nerves were divided, presented large bulbous ends and were firmly bound down in scar tissue. Figure 6 was made five months after injury. The extensive sensory changes may be noted. In this patient the Tinel sign was present and also formication in toes.

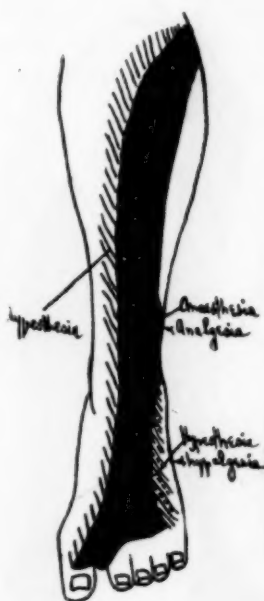


Figure 7

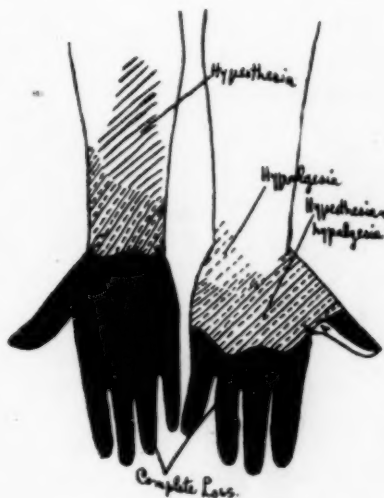


Figure 8

Fig. 7 (Case 7).—L. S., nerve injuries resulting from a gunshot wound across the popliteal space.

Fig. 8 (Case 8).—G. W., nerve injuries from high-explosive wound of the arm.

CASE 7.—L. S. received a gunshot wound across the popliteal space. Findings at operation nine months after injury: The external popliteal nerve was divided, with a large bulb at both ends.

CASE 8.—G. Wm. suffered a high-explosive wound, internal aspect of the right arm, the middle third. Findings at operation ten months after injury: The ulnar and median nerves were divided and separated for a distance of  $1\frac{1}{2}$  inches. Figure 8 was made one month earlier. Extensive sensory changes in musculospiral area, no doubt due to functional disorder, should be noted.

CASE 9.—W. T. suffered a high-explosive wound, middle of the left forearm. Findings at operation nine months later: The ulnar nerve was divided

and separated for a distance of 3 inches. Figure 9 was made one month before operation. The slight sensory changes as compared with those in other ulnar nerve sections should be noted.

CASE 10.—V. J. suffered a high-explosive wound of the right axilla. Findings at operation seven months after injury: There was a very large mass of glands enveloping the nerves just below the cords of the plexus. The median nerve was divided with a 5 cm. defect. The internal cutaneous nerve was divided with an 8 cm. defect. There was a small amount of scar tissue around the musculocutaneous and musculospiral nerves. The ulnar nerve was divided

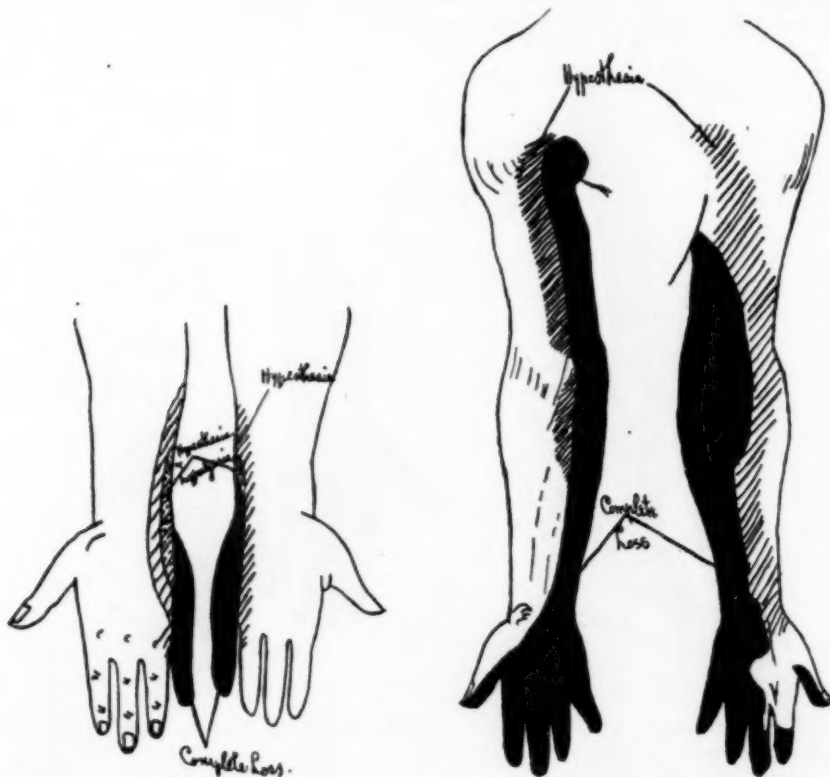


Figure 9

Figure 10

Fig. 9 (Case 9).—W. T., nerve injuries following a high-explosive wound of the forearm.

Fig. 10 (Case 10).—V. J., nerve injuries after a high-explosive wound of the right axilla.

with a 10 cm. defect. Figure 10 was made a month before operation. The patient had a positive Tinel in the median nerve and formication in the median area. The musculospiral nerve showed no changes.

All the foregoing operations were performed by Dr. Charles A. Elsberg at the U. S. Army General Hospital No. 1, Williamsbridge, N. Y.

## COMMENT

In testing for loss of sensation of touch I used a brush with long, soft hair, and it was found necessary to have the parts shaved. In testing for analgesia, I used a pin, and it was necessary to distinguish between pressure and pain. Frequently the patient would say that he felt the pressure but not the prick of the pin. In the syndrome of complete interruption anesthesia and analgesia were always present, and as a rule I found the area of the former to be more extensive, although occasionally the reverse was true. In determining the threshold stimulus and the localizing power, I employed a pair of compasses with blunt points. The results obtained were not alike in all the nerves, but were about the same in injuries of the external popliteal, sciatic and ulnar nerves.

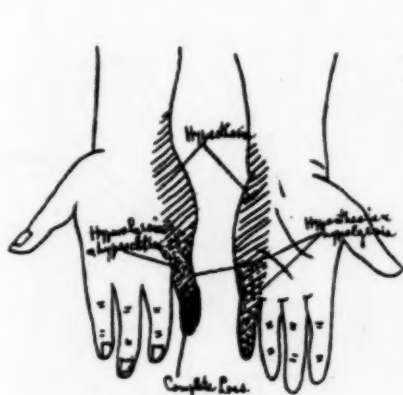


Figure 11



Figure 12

Fig. 11 (Case 11).—H. H. J., nerve injuries resulting from a high-explosive wound of the forearm.

Fig. 12 (Case 12).—L. J., nerve injuries from a high-explosive wound of the forearm.

Hypesthesia and hyperalgesia were always present in complete sections, comprising a zone about the area of complete sensory loss. On the other hand, a hypesthetic area was not always hyperalgesic, and vice versa. Examination of the figures will show that there are areas where hypesthesia was present and the sensation of pain was normal.

The sensation of heat and cold was generally abolished, but more often that of heat. Warm felt as cold, or cold as warm was a rare finding.

In the syndrome of complete interruption, areas of hyperesthesia and hyperalgesia were never encountered. Whenever such areas were found the diagnosis of complete section was doubtful.

## 2. SENSORY SIGNS OF REGENERATION

After investigating a great many cases, I came to the conclusion that the Tinel sign of regeneration possesses a corroborative value only. It was present in cases of complete nerve section with no regeneration. In Case 10 the median nerve was found to be divided, yet the sign was present. The same is true of Cases 2 and 6. I know no reason for its presence in such cases. In ulnar and median lesions it is necessary to question the patient closely as to the exact location and extent of the sign. Occasionally in ulnar lesions it was present in the median area. This could be explained either by ulnar and median

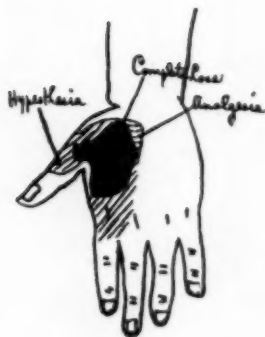


Figure 13

Fig. 13 (Case 13).—D. T., nerve injuries following a high-explosive wound of the elbow.

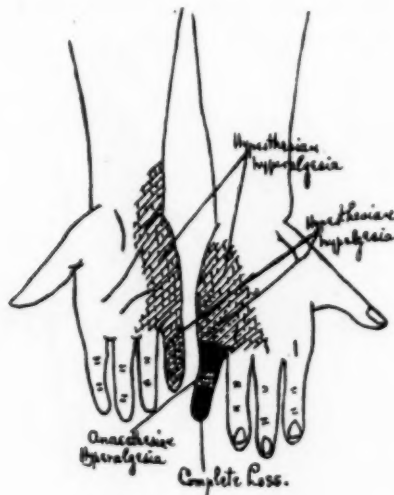


Figure 14

Fig. 14 (Case 14).—F. H., nerve injuries after a high-explosive wound of the forearm.

anastomosis, or by transmission of the pressure or tapping to the median, which had suffered a slight injury and was now regenerating.

In injuries of the sciatic nerves, when only one trunk, especially the internal popliteal, was injured, the formication was oftentimes present in the external popliteal distribution.

Examination of a number of postoperative cases and cases of spontaneous recovery revealed the fact that sensation returns before motion. A study of the figures 11-17 will show the nature of the changes in: (1) partial lesions with no operation; (2) postoperative cases; (3) cases of spontaneous recovery.

CASE 11.—H. H. J. suffered a high-explosive wound, middle third, antero-internal aspect of the left forearm. Examination eight months after injury: Syndrome of partial interruption of ulnar nerve, with signs of regeneration (Fig. 11).

CASE 12.—L. J. suffered a high-explosive wound of the postero-external surface of the left forearm, middle third. Examination 203 days after injury: Syndrome of partial interruption of the left median. The Tinel sign was present. The extent of hypesthesia over palm of hand should be noted (Fig. 12).

CASE 13.—D. T. received a high-explosive wound, internal surface of the left elbow. Examination 121 days after injury: Syndrome of partial interruption of musculospiral nerve. The Tinel sign was present. The absence of sensory changes on the palmar surface of the hand should be noted (Fig. 13).

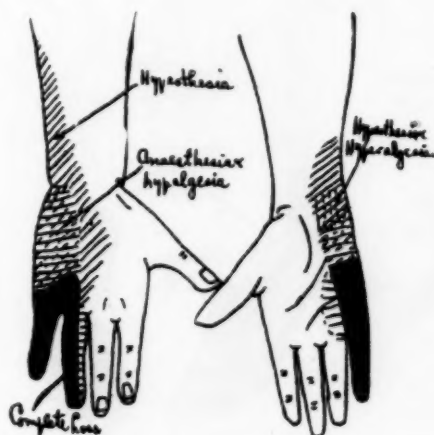


Figure 15

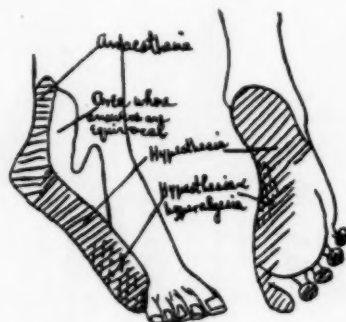


Figure 16

Fig. 15 (Case 15).—A. A., nerve injuries resulting from a high-explosive wound of the forearm.

Fig. 16 (Case 16).—C. F. W., nerve injuries from a machine-gun bullet wound of the thigh.

CASE 14.—F. H. suffered a high-explosive wound, anterior aspect, middle third of the right forearm. There was a syndrome of partial interruption of the ulnar nerve. The sensory figure was made 179 days after the injury. His history indicated that he had had also an injury to the median nerve from which he had recovered. At the last examination he had a very pronounced Tinel sign in the ulnar and median nerves. The only sensory disturbance in the median area was hyperalgesia on the upper palmar surface of the index finger (Fig. 14).

CASE 15.—A. A. had a high-explosive wound of the middle surface, internal aspect of the right forearm. The sensory figure was obtained 175 days after injury. The Tinel sign was present. There was a syndrome of partial interruption of the ulnar nerve. The area of anesthesia extended into the region supplied by the internal cutaneous nerve (Fig. 15).

CASE 16.—C. F. W. received a machine-gun bullet wound, anterior aspect of the right thigh. Examination 150 days after the injury detected a slight weakness in the distribution of the internal popliteal nerve. The Tinel sign was positive (Fig. 16).

CASE 17.—W. D. suffered a high-explosive wound of the internal surface of the upper third of the right arm. Examination 165 days after the injury disclosed in addition to a lesion of the musculospiral nerve, also a partial injury (slowly regenerating) and a musculocutaneous lesion. He had a strongly positive Tinel sign for the ulnar nerve. The musculocutaneous nerve in spite of the injury, did not show any motor loss, the only evidence of its injury having been the change in sensation.

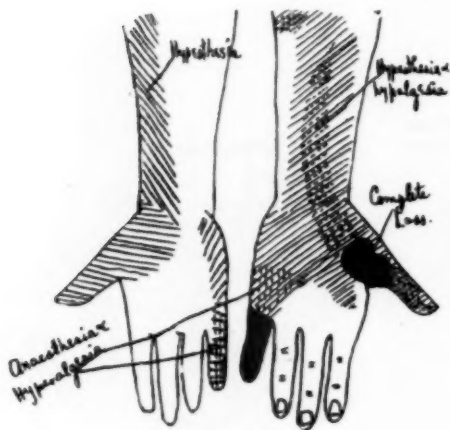


Figure 17

Fig. 17 (Case 17).—W. D., nerve injuries following a high-explosive wound of the arm.

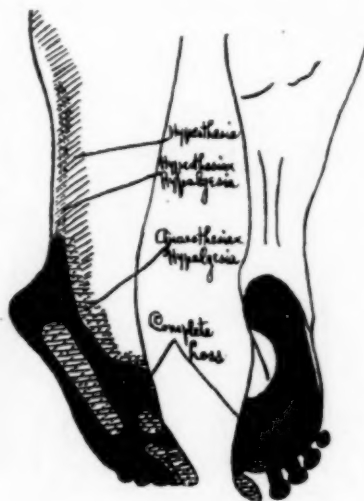


Figure 18

Fig. 18 (Case 18).—L. P., nerve injuries after gunshot wound of the thigh.

The following patients are those who were operated on either on account of complete interruption or because of imperfect regeneration:

CASE 18.—L. P. This patient has been discussed under Figure 1. The present figure was obtained three months after nerve suture. It shows beginning regeneration. In the middle of the anesthetic and analgesic area, on the external surface of the foot, a small area of hypalgesia appeared, which in course of time would become hyperalgesic. The anesthetic area also became smaller. The figure also shows the independence of the fibers for superficial sensation of touch and deep sensation of pain. There were areas in which anesthesia was present with hypalgesia, and an area with analgesia and hypesthesia. The latter area was previously anesthetic and analgesic. In spite of the improvement in sensory symptoms, there was no motor improvement (Fig. 18).

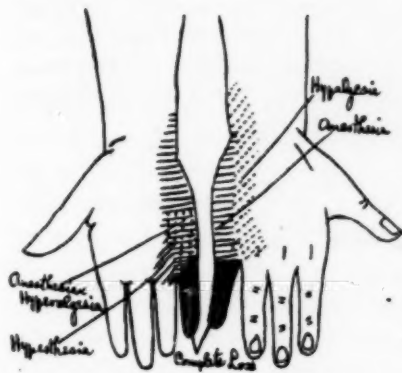


Figure 19

Fig. 19 (Case 19).—O. W., nerve injuries resulting from a high-explosive wound of the forearm.

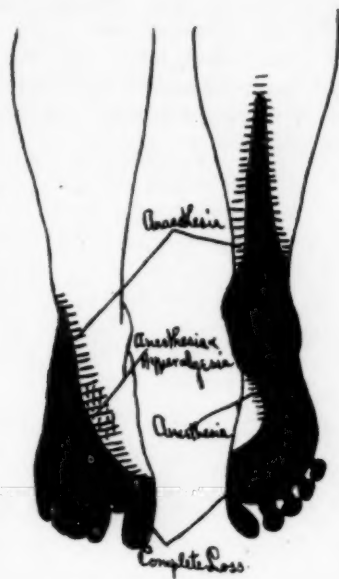


Figure 20

Fig. 20 (Case 20).—S. B., nerve injuries from a high-explosive wound of the thigh.

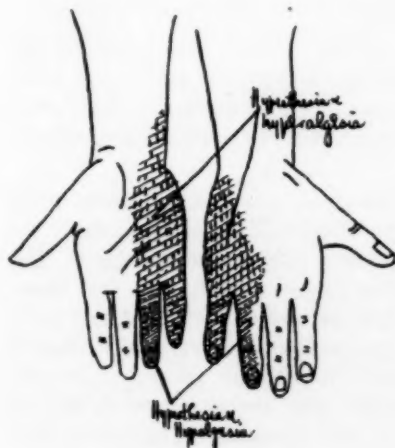


Figure 21

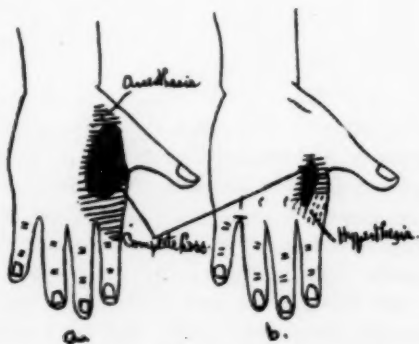


Figure 22

Fig. 21 (Case 21).—S. M., nerve injuries following a high-explosive wound of the forearm.

Fig. 22 (Case 22).—B. M., nerve injuries after a high-explosive wound of the arm. Motor improvement was more rapid than sensory.

CASE 19.—O. Wm. suffered a high-explosive wound of the internal surface of the right forearm, lower third. Examination was made ninety-three days after nerve suture for a complete ulnar. The Tinel sign was present. The improvement in motion was very slight, but sensory improvement was very marked. Muscular atrophy was very pronounced (Fig. 19).

CASE 20.—S. B. received a high-explosive wound of the posterior surface of the right thigh, middle third. The figure was obtained two months after nerve suture for a complete sciatic. There was no motor improvement but hyperalgesia and hyperesthesia had appeared. In this patient the Tinel sign was absent, and there were no signs of regeneration except the slight sensory change. He also showed very little muscular atrophy, much less than would be expected after an injury of about six months' duration (Fig. 20).



Fig. 23 (Case 23).—McT., nerve injuries resulting from a high-explosive wound in the popliteal space.

CASE 21.—S. M. had a high-explosive wound, internal aspect of the right forearm, lower third. He had a partial ulnar, regeneration not having taken place on account of mechanical obstruction. Small fragments of bone were removed. Figure 21 was obtained 173 days after operation. There was no motor improvement.

CASE 22.—B. M. This patient was the first one seen in whom motor improvement was more rapid than sensory. Figure 22 (*a* and *b*) shows the sensory disturbances before and after operation, only six weeks apart. The patient had a high-explosive wound, anterior surface of left arm, just above the elbow. A small piece of shrapnel was still present in the wound and probably interfered with regeneration of the musculospiral nerve, which showed partial injury. Figure 22*a* shows the sensory disturbances taken four months after injury. He was operated on one week after the examination, or five months after the injury, and the fragment was extracted. Thirty-nine days after operation Figure 22*b* was obtained. As can be seen, there was slight improvement in sensation; on the other hand, the improvement in motion was remarkable. All the movements of extension were possible, without limitation in the slightest degree.

COMPLETE SPONTANEOUS REGENERATION OF THE EXTERNAL  
POPLITEAL NERVE

CASE 23.—McT. had a high-explosive wound in the right popliteal space. Examination seven months after injury disclosed a slight limitation in the dorso-flexion of the right foot and a suggestion of right toe drop. Examination of flexors and extensors of the foot showed no abnormality. Figure shows sensory disturbances (Fig. 23).

## COMMENT

The first sensory changes appeared in my cases as early as thirty days after operation or after injury. In cases of spontaneous recovery it is impossible to say when the first changes appear. The musculospiral and the ulnar probably regenerate earlier than any other nerve. The first sign of regeneration of the sciatic nerve appeared as early as two months after operation.

The second phase was the reduction of the anesthetic and hypesthetic areas. Especially the anesthetic area became smaller and spots of hypesthesia appeared in the middle of the anesthetic zone. With the reduction of the analgesia there appeared patches of hyperalgesia. These become more marked in the course of time, until anesthesia and analgesia entirely disappeared.

It is important to test for vibration perception in such cases with the tuning fork. Hyperesthesia was not so frequently found as hyperalgesia. During the third phase of regeneration, analgesia and anesthesia disappeared leaving a small area of hypesthesia or hyperalgesia. Hyperesthesia and hyperalgesia are never present in complete interruption.

I am especially indebted to Major G. E. Price, M. C.; Major H. O. Feiss, M. C.; Lieut.-Col. E. G. Zabriskie, M. C., and to Dr. (formerly Major) T. H. Weisenburg for opportunities, stimulus and advice.

## Abstracts from Current Literature

PATHOGENESIS OF DIPHTHERITIC PARALYSIS. F. M. R. WALSH, Quart. J. Med. **11**:191 (April) 1918; **12**:14 (Oct.) 1918; **12**:32 (Jan.) 1919.

This interesting paper, in three parts, first reviews the rather scant literature on the subject, then compares the mode of invasion of the nervous system in diphtheria to that in rabies, details the author's unusual experience in thirty cases of paralysis following extrafaucial diphtheria, and finally draws certain conclusions.

The author's cases were those of diphtheritic invasion of wounds and cases of so-called "barcoo rot" or "septic sores" which he shows may fairly be considered diphtheritic, although until recently their nature was thought to be obscure. His conclusions are:

1. Palatal paralysis does not occur except after faucial diphtheria.
2. The musculature of the palate and the region of the infective focus derive their innervation from the same source, the glosso-pharyngeal-vagus-accessorius nuclear system and its peripheral fibers, and are thus closely related anatomically.
3. Similarly, in extra-faucial diphtheria the paralysis often shows (27 per cent.) an onset anatomically related to the infective focus.
4. Polyneuritis follows both faucial and extra-faucial diphtheria equally, and irrespective of the site of the infective focus presents a remarkably constant symptom-complex.
5. Paralysis of accommodation also follows both forms of infection, though the faucial more frequently (86 per cent.) than the extra-faucial (33 per cent.). It is, therefore, not a "local" paralysis, and its more constant association with faucial infections may be explained by the close proximity of the local central nervous lesion in this case to the oculomotor nuclei. To this extent a "local" factor may be admitted.
6. The grouping of the symptoms of tetanus, adopted by Meyer and Ransom, into local, specific, and generalized, may be applied equally well to the nervous phenomena of diphtheria. The palatal paralysis constitutes the "local," the ocular affection the "specific," and the polyneuritis the "generalized" forms of diphtheritic paralysis. In extra-faucial diphtheria, as in tetanus, the "local" paralysis varies with the site of the infective focus.
7. As for the pathologic processes underlying this syndrome, it seems highly probable that as regards the local paralysis we are dealing with an "ascending lymphogenous toxi-infection" (by the perineural lymphatics) of the central nervous system from the infective focus. The essential pathologic lesion here is central and not peripheral, and is situated in the nuclear complex already described.
8. The ocular and generalized symptoms are probably the result of the circulation of the toxin in the blood stream, whence it gains access to the whole nervous system, central and peripheral. The essential nervous lesion here is probably both central and peripheral.

Dr. Walshe states that these conclusions require confirmation by experimental and pathologic investigations. In part three, however, he details a case of diphtheritic ulcer of the perineum which strongly supports his conclusions. This sore was followed first by anesthesia of perineum, anus and adjacent parts of buttocks and thighs, then by involvement of bladder and later by multiple neuritis. The anesthesia of perineum and thighs showed the ordinary saddle shape of sacral cord disease and the author thinks it was due to involvement of the second, third, fourth and fifth sacral segments.

PATRICK, Chicago.

THE OPERATIVE TREATMENT OF SPASTIC PARALYSIS (HEMI-  
PLEGIA, MONOPLÉGIA, PARAPLEGIA) IN GUNSHOT WOUNDS  
OF HEAD AND SPINAL CORD. PROF. FOERSTER, Breslau, Deutsch.  
Ztschr. f. Nervenhe. May 7, 1918.

The author describes the procedures followed in sixteen cases of spastic paralysis of various types following gunshot wounds of brain or cord.

The operation of choice in severe spastic paraplegia is resection of the posterior roots. This was done in two cases of this type according to the following technic: The dura was exposed from the eleventh dorsal spine to the second lumbar vertebra, with resection of two thirds of the posterior roots in the region of the lumbo-sacral enlargement. In the second case, the dura was exposed at the first operation and opened eleven days later with resection of two thirds of the posterior roots as in the first case.

In this exposure the roots are found lying closely together allowing approach through a comparatively small opening in the bony covering.

Not only was the spasticity completely and permanently removed, but the voluntary movement to a large extent returned.

In one case of severe paraplegia with spastic paralysis of both arms due to a wound of the head, resection of the posterior lumbar roots and the second sacral roots was followed by relief of spasticity, but voluntary movement was little improved owing, Foerster states, to the fact that the motor centers were destroyed at the time of the accident.

Not all paraplegias following injury to the uppermost part of both central convolutions are as severe as the cases noted above. The paralysis and spasticity may involve only both feet, while knee and hip joints are either entirely free or are only slightly spastic-paretic. The lesion is, in these cases, in the inner side of the right and left paracentral lobule. Here lies the center for the dorsal and plantar flexion of the foot while the center for the muscles moving the knee and hip joints is to be sought on the convexity of the same convolution. Several times spastic paralysis of both feet was associated with ataxia of both legs. In these cases the position sense in both legs was completely lost, the central lesion extending backward on both sides to the region of the parietal convolutions.

For spastic contracture affecting single muscle groups plastic operations on the tendons is to be recommended, care being taken not to injure the tendon sheath and synovial channels. Beside the tendo-achillis, lengthening of tendons may be done successfully on the flexor carpi radialis, palmaris longus, flexor carpi ulnaris, on the biceps flexor cruris, semi-membranosus and semi-tendinosus.

In muscles where a tendinous attachment is wanting, Stoffel's operation on the peripheral nerves may be used. The latter method is especially useful in spasticity affecting the quadriceps extensor and adductors in the lower extrem-

ity and the pronators, biceps, coraco-brachialis, brachialis anticus, etc., in the upper extremity.

In the long flexors of the fingers the Stoffel operation is to be recommended as simpler than the attack on the numerous tendons. The same holds true for the flexors of the toes. With the relief of the spasticity in the operated muscles voluntary movement returns in the antagonists.

Unfortunately, by this method the source of the spasticity is not removed; the centripetal impulses continue so that in severe spastic conditions there is a return of the condition. For a permanent result so many motor fibers must be resected that paresis results.

The following commonly occurring types of contracture deserve special consideration:

1. Contracture of the plantar flexors of the foot and the associated paralysis of the dorsal flexors.
2. The inclination of the foot to assume a position of supination and thereby to combine, under certain circumstances, with a marked inversion of the outer border during the supporting phase.
3. The "clawing in" of the toes in raising the foot and during the supporting stage.
4. Spastic contracture of the quadriceps and the associated paralysis of the flexors of the knee.

1. For the relief of spastic contraction of the plantar flexors of the foot lengthening of the tendo-achillis is given the first place. The entire sole of the foot rests on the ground, the gait improves and the power of voluntary movement returns in the dorsal flexors.

2. For the relief of the inclination of the foot to supinate during the attempt at dorsal flexion one of two methods may be employed. (a) The tibialis posticus may be partly paralyzed by resecting the nerve bundle to this muscle in the popliteal space. One quarter of the fibers are allowed to remain. (b) The tendon of the tibialis anticus may be split in its entire length, the separated portion being transplanted into the outer border of the foot.

3. To combat the tendency of the toes to become forcibly flexed when the foot is raised or during the supporting phase, the nerve bundle to the flexor longus digitorum is resected in the neighborhood of the posterior tibial nerve. In many cases with secondary retraction of the toe flexors tenotomy of the flexor tendons may be resorted to.

4. For the relief of spastic contracture of the quadriceps extensor femoris exposure of the femoral nerve and resection of a part of the bundle supplying each head of the muscle is recommended.

In general, there is found in the hemiplegic arm spastic contracture of the flexors of the fingers and a corresponding paralysis of voluntary movement in the extensors. In the thumb there exists contracture of the long flexors and the adductors with a corresponding paralysis of the extensors, abductor and opponens. In the wrist, in the majority of cases, there is a spastic contracture of the flexors and paralysis of the extensors. At the elbow, spastic contraction of the flexors and paralysis of the extensors is the rule. At the shoulder there is found spastic contracture of the latissimus dorsi and pectoralis major and paralysis of the elevators of the arm. The inward rotators are spastic and the outward rotators paretic.

The operative measures for the relief of these disturbances are as follows: The spastic contracture of the flexors of the fingers is best relieved by partial

resection of the fasciculi to the flexor profundus digitorum and flexor sublimis digitorum in the neighborhood of the median and ulnar nerves. At most only one third of the fibers are incised and then only after the fasciculi have been extensively split up so that the resection may not involve too many fibers going to any one tendon. The flexors of the thumb are dealt with in the same manner. Sometimes, to improve abduction the tendon of the flexor carpi radialis is transplanted into the tendon of the extensor brevis pollicis.

Adduction contracture of the thumb may be treated either by resection, in the palm of the hand, of the nerve to the adductor pollicis or by tenotomy of the insertion fibers of the adductor.

The spastic contracture of the flexors of the hand is best relieved by plastic lengthening of the palmaris longus and flexor carpi radialis. Care must be taken not to make the operation too extensive lest the contractile power be weakened. Their action is necessary for fixation of the wrist during the act of extending the fingers.

The nerve to the pronator radii teres is resected as it leaves the median nerve for relief of pronatory spasm. Resection of one third of the musculocutaneous nerve is done for the relief of contracture of the flexors of the forearm.

For contracture of the latissimus dorsi and pectoralis major muscles, Foerster formerly resorted to plastic lengthening of the respective tendons, but in order that the operation should be extensive enough it was necessary to invade the muscle tissue thereby leading, in most cases, to weakening of the muscles. He now exposes the brachial plexus and resects the nerves to these muscles.

Tenotomy of the subscapularis tendon for spasm of this muscle has also given way to partial resection of the subscapular nerves as they leave the brachial plexus.

The article is illustrated by photographs of cases operated on and by descriptive case histories.

INMAN, San Francisco.

DISMORFIE ENDOCRINE. F. GIANNULI, Riv. di antropol. 21:215.

The morphologic type of the individual and his deviation from the normal forms the basis on which the Italian school has oriented the study of endocrinology. From the clinical morphology started by De Giovanni a few decades ago, the author says, we have passed to the endocrine morphology which opens the horizon toward the origin of diseases and which should constitute an essential preparation to the modern clinic.

The author classifies the endocrine deformities into three great categories, namely, deformities of monoglandular type, of pluriglandular type and of dysglandular type. He observes that pure monoglandular types are not the cases encountered in practice. Two cases illustrating the first category and one case illustrating the third type are reported. The first two correspond to the fundamental types of eunuchs differentiated by Tandler and Gross, namely, the eunuchus gigas and the eunuchus lardaceus. The third is a eunuchoid in which the masculine sex is prevailing. The author points out that the same two types of eunuchs consecutive to extirpation of the sex glands may be found in primary diseases of the hypophysis. This fact indicates and proves the existence of intimate correlations between these two endocrine systems, correlations which have a great value in the genic functions and which give to the hypophysis the same biologic importance ascribed to the sex glands.

CASE 1.—The characteristics of the first specimen, a man, 36 years of age were:

The height was 1.98 meters (6 feet 6 inches). The length of the lower extremities was 1.3 meters (4 feet 3 inches). There were thoracic deformities, the pelvis being large, almost feminine. There was complete absence of hair from the face, from the mons veneris and from the entire body. The penis, scrotum and testes were rudimentary and presented a rudimentary vulva opening. The voice was of the feminine type. Mentally the patient presents the hebephrenic type of dementia praecox and shows a tendency to passive pederasty.

CASE 2.—The second subject, a woman 19 years old, suffering from a chronic hydrocephalus, combined in herself three syndromes: one neurologic, one psychic and one endocrine—a clinical picture extremely rare. She menstruated at the age of 12, then the symptoms grew gradually worse. At the age of 14 a tetraparesis appeared together with a menopause praecocissima (as the author calls the early disappearance of the menstruation in this case). Since then the growth of the limbs stopped, which fact was accompanied by a rapid and abundant increase of fat. The physical impotence, which reduced the patient's life to a purely vegetative one, was associated with a marked mental deterioration which culminated in apathetic dementia. The main morphologic stigmata are: somatic infantilism, small hands and feet not proportional to the length of the limbs, fingers hammer shaped bilaterally; absence of hair from the body and from the pudenda which shows a very elementary constitution characterized by the absence of the labia minora and of the clitoris; vagina infantile.

The author, after illustrating these two fundamental types, makes a rapid review of the experimental and clinical work carried out on the sex glands and hypophysis by Pellican, Tandler and Gross, Erdheim and Stumme, Loris, Pende, Goldstein, Peritz, and others, and discusses the different theories on the correlations existing between these two endocrine systems. He admits that although the general correlations seem to be sufficiently proved, yet the specific ones are not proved, so we are unable to explain the physiologic and the physiopathologic mechanism operating in the interdependence existing between the hypophysis, its lobes, and the sex glands. Except in cases of cerebral diseases it is very difficult to determine which is the gland primarily involved in the case of a eunuch. Of the two cases reported by the author, the primary involvement of the pituitary may be clearly seen in the second case, while the pathogenic origin cannot be stated in the first case.

The author thinks that the first case was due to the primary lesion of the sex glands and expresses the opinion that the giantism is secondary to morbid processes of the testes while the primary lesion of the hypophysis leads to acromegaly.

After emphasizing the importance of the morphologic study of the subject as a necessary preparation to the modern clinic, the author advances the following conclusions:

1. The endocrine deformities best defined by experiment and by clinical experience are those which produce the type of the eunuchus gigas and that of the eunuchus lardaceus of Tandler and Gross.
2. There are eunuchs and eunuchoids originating from pituitary disturbances just as there are from experimental or pathologic castration in both sexes.

3. The pictures of the pituitary and gonadic eunuchoidism often are alike in the semeiologic effects. This fact proves that between the hypophysis and the sex glands exists a system of intimate endocrine relations which govern the sexual life and the skeletal development.

4. This important endocrine system governs the genetic function and the physiologic evolutional and involucional phases of the sex glands and also the diseases of the sexual apparatus have a histologic repercussion over it.

5. As several endocrine glands comprise the genesic endocrine system the eunuchoid deformities may be either chiefly monoglandular or pluriglandular in type.

6. As the genesic endocrine system may be subject to pathologic changes which manifest themselves somatically not with functional quantitative disorders but with qualitative disorders, there may be some aberrant eunuchoid deformities which present only vague analogies in monoglandular eunuchs and which belong to a third group of eunuchs, the dysglandular eunuchs.

SANTE NACCARATI, New York.

THE PROBLEM OF FATIGUE. R. A. SPAETHE, PH.D., *J. Indus. Hygiene* 1:22 (May) 1919.

This article is a comprehensive review of the literature on fatigue, the various phases of the subject being taken up in turn. Part 1 deals with the theories of fatigue, physiologic and psychologic. The author first summarizes concisely the chemical point of view, that the problem is one of energy transformation with a resulting "dynamic equilibrium;" that is to say, work that reduces the available energy to low potential, is equalized by rest, which restores it to a high potential. The results of experimentation on the isolated muscle are also discussed and the theoretical "dynamic equilibrium" is correlated with the experimental finding of a "fatigue level." This "fatigue level" is attained when muscular work is carried on at such a rate that the metabolic products, such as lactic acid, are removed from the muscle at the same rate as energy is brought to the muscle in the form of nourishment. These points are well illustrated by Figures 2, 3 and 4 (but Figures 3 and 4 should be more thoroughly explained, and Figure 3 should have an arrow showing the direction of the kymographic tracing). Sherrington's theory that fatigue is localized in the synapse is clearly put forward by a quotation from Ash, and the fact that "nervous activity appears to precede in an astonishingly economical fashion" is discussed.

The most interesting part of the paper to the neuropsychiatrist is the exposition of the theory of the "varying energy level." This is a conception long needed in physiology and medicine, and though promulgated in terms of introspective psychology by James and McDougall, it now seems at last to be brought into a working relationship with physiology and pathology. Cannon's work is cited to show that the adrenals may be looked on as the physiologic basis for such psychologic concessions as James' "reservoirs of power." The importance of the emotions is discussed at length, and it is shown that emotive stimuli by releasing epinephrin may "carry the human organism through critical periods," but that "if we draw too deeply and continuously on our reservoirs of energy we may develop a condition of hyperesthesia and ultimately suffer a complete nervous breakdown."

It is most gratifying to see the emotions given their full responsibility as a cause of nervous breakdown, and by such straightforward mechanistic theories only can we lay a basis for a dynamic psychology; but when the author

states that—"from this point of view, nervous breakdown would really be a condition of adrenin (epinephrin) poisoning"—we must take issue with him. It is probable that certain kinds of breakdowns do occur commonly in this way, especially the conditions of hyperesthesia and the anxiety states arising from environmental conditions (such as war), but "nervous breakdown" is a broad term and it must not be forgotten that many breakdowns have as their outstanding features lack of emotion, energy and satisfaction. (And just such cases as these have gone to war and thrived on the abundant stimuli of the new environment.)

In the second part of the paper the tests for fatigue are discussed, and it is shown that no one test can be satisfactory because of the complex nature of fatigue. The relation of fatigue to efficiency is shown to be less simple than most statisticians believe. Here again the "emotive stimulus" is given its proper value. The war experiences in speeding up industrial output are cited as cases in point. Here strong emotional stimuli were used over long periods and resulted in some breakdowns and in a widespread industrial unrest. In the author's words: "The problem of industrial fatigue boils down then, to the practical question of detecting the signs of incipient psychoneuroses." It is, however, important to understand that industrial fatigue does not arise from simple overwork but from returning to the job day after day, having *rested insufficiently* between the hours of work. Thus industrial psychology comes back to the problem of the individual. What a pity that Carleton Parker could not have lived to see his theories taken up by the biologists.

Examples of increased output following the shortening of working hours are given, and "scientific management" is justly criticized. The paper ends with a bibliography of eleven pages, showing with what painstaking care the author compiled his facts.

COBB, Boston.

PITUITARY DISTURBANCE FROM ADENOIDS. P. CALICETI, *Pediatrics* **27**:161 (March) 1919.

Caliceti recalls the relationship of the pituitary to the adenoids and the damage that may be done to it by the latter. He reports two cases that bring out these facts and that show quite forcibly the great importance of early removal of the adenoids before damage has been done to the pituitary, followed by the well-known changes in body and mind. Beside the operative procedure, he finds pituitary feeding beneficial.

FRANTZ, New York.

SUGAR TESTS IN HYPERTHYROIDISM AND OTHER ENDOCRINAL DISORDERS. JOHN R. WILLIAMS and ELEANOR M. HUMPHREYS, *Arch. Int. Med.* **23**:537 (May) 1919.

These authors state that the glucose tolerance and utilization test proposed by Hamman and Hirschman (*Arch. Int. Med.* **20**:761 [Sept.] 1917) and as later modified by Janney (*J. A. M. A.* **70**:1131 [April 20] 1918), is a useful procedure to differentiate those metabolic disorders in which traces of a reducing substance are excreted in the urine. This test is superior to others that depend solely on the determination of urine sugar as a means to measure the degree of disturbance in carbohydrate metabolism in hyperthyroidism and other endocrinal disorders.

ARMSTRONG, Katonah, N. Y.

CEREBRAL HEMORRHAGE OF THE NEW-BORN. MARGARET WARWICK,  
Am. J. M. Sc. **158**:95 (July) 1919.

This paper is based on a study of necropsy findings. Little has been written on this subject since 1885, at which time Sarah McNutt established the relationship between cerebral hemorrhage at birth and Little's disease.

The study of necropsies reveals that intracranial hemorrhage is found in from 12 to 32 per cent. of cases. The location varies, the hemorrhage being subdural, subarachnoidal, intracerebral, intraventricular, etc. The most frequent location is over the cerebrum because the veins in this area are afforded little protection, and in rapid labor they have little chance to mold themselves and are ruptured by the over-riding of the parietal bones.

The author states that punctate hemorrhages of the epicardium and parietal pleura are always found associated with intracranial hemorrhage, and in many instances there is a general hemorrhagic diathesis. Partial atelectasis of the lung is almost always associated with cerebral hemorrhage which might be a contributory and not a primary condition. There is usually hemorrhage without softening of the brain substance, death usually occurring before these changes take place.

The causes of cerebral hemorrhage of the new-born, as given by many authors, are:

(a) Long interval of time between birth of body and birth of head. (b) Hemorrhage following asphyxia caused by twisting of the cord about the neck. (c) Too great pressure of perineum. (d) Severe labor pains causing pressure on the sides of head, which increases long axis and presses brain forcibly against the tentorium tearing its radiating fibers. (e) Incomplete or too quick molding resulting in rupture of veins.

The author after discussing these theories and study of individual cases concludes that there are three primary causes: (1) Traumatism of all kinds at birth; (2) congestion or stasis with rupture of vessels, that is, due to malpresentation, overgrowth of child, twins, etc.; (3) diseased condition of child in intra-uterine life with no relationship to labor, that is, hemorrhagic diseases of the new-born, prematurity, syphilis, congenital heart diseases and other toxemias.

Cerebral hemorrhage of the new-born is not brought about by one single cause but by an interrelation and interaction of a varying number of causes found in the complex circumstances governing labor.

DELONG, Philadelphia.

## Society Transactions

### NEW YORK NEUROLOGICAL SOCIETY

*Three Hundred and Seventy-Third Regular Meeting, held at the  
Academy of Medicine, May 6, 1919*

WALTER TIMME, M.D., *President*

#### PRESENTATION OF CLINICAL MATERIAL. By DR. I. ABRAHAMSON.

Dr. Abrahamson presented six cases recovered from epidemic polioencephalitis and showed several photographs of the patients taken during the active stage of the disease. They all gave a history of epidemic influenza a short time before the development of the polioencephalitis.

CASE 1.—Present illness dated back five weeks. Patient showed Parkinson face, attitude and gait, typical tremor of paralysis agitans and cogwheel phenomenon. The condition had recently decidedly improved.

CASE 2.—The status showed a typical Parkinson face, attitude and gait, tremor mainly on intention, cogwheel phenomenon, rigidity of the extremities and bilateral facial weakness. There had been marked and constant improvement.

CASE 3.—The patient had had epidemic polioencephalitis one month ago. There was a lack of facial mobility.

CASE 4.—Four weeks ago the patient had encephalitis. He left the hospital almost well, but recently his head commenced to move involuntarily, breathing felt hampered, head and neck felt rigid, head bent forward to the right, and there was a beginning masklike face and attitude of a Parkinson. The Wassermann test of blood and spinal fluid was negative.

CASE 5.—Six weeks ago the patient had influenza. Since that time he had the following symptoms: dizziness, tinnitus, altered speech diplopia, drowsiness, mental confusion, confabulation, spasmodic cough, and hoarseness. There was present an external ophthalmoplegia, partial drowsiness, deviation of tongue to the right, weakness of right face and tremors. He had grown very stout since the onset of the illness. There was marked bulimia, polyuria, polydipsia, loss of ejaculatory power and impairment of taste. There had been a slow improvement.

CASE 6.—Initial symptoms which appeared in January, 1919, were headache, vertigo, gastric distress, weakness of extremities, especially the left, herpes labialis, fever; at first he was languid, then somnolent, eyes staring and pains in the extremities. There developed in the left leg a loss of control, then a tremor and then control of the left arm was lost and tremor developed. Following this his face became masklike, shoulder swing in walking was lost, he perspired freely, lost weight, there was difficulty in turning around quickly, retropulsion, marked slowness of all voluntary movements and twitching of right toes. On examination he presented all the signs of a typical Parkinson syndrome: masklike face, attitude and gait, tremor, rigidity and lack of associated movements. The course was progressive at first, but recently there had been steady improvement.

DR. C. C. BELING, Newark, presented a recovered case of epidemic encephalitis which showed the following sequelae: a general Parkinsonian attitude without muscular rigidity; fibrillary tremors of the tongue, face and lips; a high-pitched voice; drooling of saliva; slowness and monotony of speech; paralysis of accommodation of ocular muscles; and double facial palsy, more marked on the right. The patient had suffered from epidemic influenza and pneumonia in October, 1918, from which he apparently recovered. On March 4 he again had influenzal symptoms, followed suddenly by diplopia two days later. On March 8 he developed a spontaneous nystagmus on both sides, more marked on looking toward the right. On March 10 he became somnolent and lethargic and went into a stuporous, semiconscious condition which continued for about three weeks. During this period several lumbar punctures showed normal cerebrospinal fluid but under increased pressure. On March 29 the outstanding symptoms were a cerebellar attitude; bilateral facial paralysis, more marked on the left; increased knee reflexes, the right more than the left; tendency to hyperextension of big toes on stimulation of the soles, more marked in the right; slight clonic movements in the left ankle and a well-marked Oppenheim reflex on the right; numerous irregular movements of the eyeballs without any definite isolated nerve palsy; slight ectopia of the pupils to the inner sides; abdominal reflexes absent except for a slight left epigastric response; marked retardation of speech; general motility and anterograde amnesia for events of the previous two weeks.

CLINICAL EXPERIENCES WITH EPIDEMIC CENTRAL OR BASILAR ENCEPHALITIS. Presented by DR. B. SACHS.

This paper was based on his experience with about thirty cases at Mount Sinai Hospital and fourteen cases seen in consultation. There were only three fatalities in the former group which was not a high percentage, but out of the fourteen cases, five died with symptoms of marked bulbar involvement which made the prognosis in these cases ominous.

The term lethargic encephalitis was ill advised; it was not the encephalitis but the patient who was lethargic, and if one considered the predominance of central or basilar symptoms, a more appropriate name was not far to seek. The clinical symptoms were very striking; broadly speaking, after a brief period of drowsiness, headache, vertigo and general malaise, the patient passed into a state of lethargy associated with symptoms pointing to involvement of the cranial nerves. Meningeal symptoms were not obtrusive. The ptosis, ophthalmoplegia externa of the nuclear type, abducens paralysis, facial palsies, which were often double, cerebellar attitude of the head, difficulties of phonation and deglutition, fibrillary tremors of the tongue, double spastic paraplegias, all suggested an encephalitis that might involve the brain stem from the larger ganglions to the pons and medulla oblongata. In addition to these symptoms, the forced attitudes, catatoniac states, occasional impulsive laughter, masklike expression of countenance, even atrophies of the interossei muscles, led to the inference that the anatomic processes might involve the thalamus at one end and the cervical ganglionic cells at the other.

While awaiting definite proof of the nature of the virus, a study of which was being conducted by Strauss, Hirshfield and Loewe, one might lay stress on the fact that in many of the cases, and in the majority of those seen in consultation practice, there had been a distinct history of influenza preceding the onset of the lethargic disorder by several weeks. If this sequence was not a mere coincidence, this epidemic encephalitis bore a resemblance to the

postdiphtheritic palsies in its occurrence weeks after the initial infection. If this was a postinfluenzal infection, it was curious that previous epidemics of influenza had not been followed more frequently by similar disorders. The condition was so different from anything that had occurred before that it was impossible that it should not have been noted. The stupor was not the ordinary kind; the patient lay inert with closed eyes and expressionless face, but was apparently aware of what was going on about him and readily responded by nodding to questions that were put to him in a low tone of voice.

There was as yet no proof that the course of the disease was influenced by treatment, but it was a fact that the cases that recovered had received careful nursing and feeding. The treatment was eliminative and purely symptomatic in the absence of a specific remedy.

The prognosis of the disease was determined largely by the site of the lesion. All the fatal cases in this series had been bulbar forms. The patients had not succumbed to the toxicity of the disease but from the fact that the cardiac and respiratory centers had been the site of predilection.

Many authors had been inclined to note a resemblance between the poliomyelitis virus and the virus of epidemic encephalitis, but it was a point to remember that there was extreme toxicity and rapid development of the disease in fatal cases of poliomyelitis, while the fatal cases of epidemic encephalitis ran a course lasting from one to seven and eight weeks. The laboratory and experimental investigation would have to furnish conclusive evidence on this point.

#### REPORT OF THE COMMITTEE ON EPIDEMIC POLIOENCEPHALITIS (LETHARGIC ENCEPHALITIS).

Dr. I. Abrahamson, chairman of the committee, read this report which embodied the number of cases and seasonal incidence of all known epidemics of this disease up to the present time. An important difference between the epidemics in foreign countries and in the United States lay in the fact that the lymphocytosis so common here had been the exception abroad. Various names had been given to this disease: epidemic encephalitis, lethargic encephalitis, influenzal encephalitis, pontobulbar encephalitis, epidemic stupor, epidemic botulism, etc. The name polioencephalitis was preferable for three reasons: The brain was mainly affected in most cases, the nuclear involvement dominated the pathologic picture, and it was a companion disease to poliomyelitis. It was recommended that the term lethargic be dropped principally because lethargy characterized only a minority of the cases. It was pathologically and experimentally established that epidemic polioencephalitis was a distinct disease and one which until recently had escaped differentiation and recognition. Reasons were cited which would seem to indicate that there was no direct relationship between influenza and epidemic polioencephalitis. The differential diagnosis between epidemic poliomyelitis and epidemic polioencephalitis was equally demonstrable. As to the clinical manifestations of epidemic polioencephalitis, syndromes of all kinds could be established. This was an infectious encephalitis, the infection originating in the nasopharynx, proceeding by the lymph streams to the basilar cerebral vessels, and then spreading through the brain stem, ganglions and cortex and also involving the upper cord, the meninges and the nerves. Those portions of the brain immediately supplied by the cerebral and basilar arteries suffered first and most severely in the majority of cases.

## DISCUSSION

DR. M. NEUSTAEDTER suggested that the best way to differentiate epidemic encephalitis from poliomyelitis would be to make the poliomyelitis neutralization test. A mixture of a 1:10 serum of a convalescent patient and true poliomyelitis virus injected into a monkey would certainly prove whether the condition were poliomyelitis or not. If the disease was poliomyelitis the serum ought to neutralize promptly the virus and the animal remain well. This suggestion was made in view of the report by the reader of the paper that encephalitis had been produced in monkeys by injecting them with a filtered suspension of the scrapings of the nasopharynx.

DR. SIMON ROTHENBERG thought that the most interesting phase of this disease was the variety of neurological syndromes that these cases presented. Although he had had eighteen or twenty cases, very few had shown identically the same picture. One simulated meningitis, another polioencephalitis with double facial palsy, a third was a cerebellar case, a fourth was of the dys-synergic cerebellar type of Hunt, a fifth was blind and showed marked coreiform movements, and finally, two other cases developed a picture of encephalitis during an attack of influenzal pneumonia. These last two cases were seen at the hospital where the first symptoms of the encephalitis were observed. In these cases the Pfeiffer bacilli were found in a culture from the throat, showing what was believed to be a connecting link between influenza and encephalitis.

DR. SMITH ELY JELLIFE recalled a family in which three patients were taken ill, apparently with influenza—one with influenzal pneumonia, who died, another with herpes and the third with a typical mesencephalitic affection. That incident had been duplicated three times in his experience and it would be interesting if it could be shown that another type of encephalitis had now been isolated which could be differentiated from an influenzal encephalitis. The first case of lethargic encephalitis he had seen was in 1890, and it would be fascinating if a new type of encephalitis might be differentiated from the others, pathologically differentiated, as this might enable still further the separation of types. From the year 1400 on, writers had accentuated the extreme variability of the different epidemic clinical manifestations which seemed to follow what were apparently clearly defined influenzal forms. It would be a great advance if one could get findings that would enable the separation of a new type producing mesencephalic manifestations.

DR. J. ARTHUR BOOTH said that during the influenza epidemic of 1889 he had seen two cases complicated by eye symptoms, in both of which there was an ophthalmoplegia externa, there being a bilateral ptosis and a paresis of the external recti muscles. There was an entire absence of undue somnolence and lethargy. In contrast with these he had seen three patients during the past winter in whom, with almost exactly the same eye conditions, there was the symptom of lethargy, and in one, a marked catatonic condition.

DR. WILLIAM M. LESZYNSKY referred to six patients that he had seen: One died at the end of two weeks; another was a physician whose particular symptom was twitching of the muscles in the extremities, but he made a complete recovery. Out of this small series only one died, all the others completely recovering. He had seen two or three cases which had been difficult to differentiate at first from the catatonic type of dementia praecox. In most of the cases gripe had occurred within a month previously.

DR. ELBERT M. SOMERS, Brooklyn, referred to the reports of forty cases called central neuritis in the 1908 and 1909 issues of the New York State Hospital's Bulletin. These cases had presented various stuporous states, muscular tension, jactitations and focal and mental symptoms. They appeared following grip, cancer, tuberculosis and infective exhaustive conditions. Thereafter, central neuritis was accepted as a cause of death by the health department of the state.

#### OBSERVATIONS ON GUNSHOT INJURIES OF THE HEAD. MAJOR

KARL WINFIELD NEY, M. C., U. S. Army, who was senior officer of the Neurosurgical Unit No. 1, A. E. F. in France, read this paper.

He emphasized the value of several procedures which experience in military cranial surgery had shown to be the cause of the immense reduction of mortality statistics. The necessity for early surgical intervention had been as pronounced in the cranial field as in others, and primary suture was the ideal treatment of all wounds. It had been found possible to effect this if all devitalized tissue could be excised before infection became established, and not only this, but certain wounds could be closed even when it was not possible to practice complete excision, and it was in this class that gunshot wounds of the brain had been placed. The surprising absence of sequelae—meningitis, brain abscess, hernia cerebri, brain fungus, etc., in these cases was most significant.

The two surgical principles of profound importance in this war, early and complete excision, gradually having found their places in the treatment of cranial injuries in so far as scalp and skull were concerned, the problem that then presented itself related to the removal of devitalized brain tissue. It was solved by Col. Harvey Cushing who combined the two principles just mentioned, and removed the disorganized brain substance by catheter suction and irrigation, completing the operation by primary closure of the dura and scalp.

In the endeavor to do a speedy operation, in the early part of the war, the procedure was often incomplete and frequently subdural adhesions were torn and the subarachnoid space opened to infection. The necessity for speed was due to the profound effects of general anesthesia on these septic patients, and realizing this the speaker became convinced that the same operative procedures were possible under local anesthesia. With the use of the same he succeeded in avoiding the shock associated with general anesthesia and was able to pursue a more deliberate operative technic without pain to, or interference from the patient, thereby insuring greater gentleness in manipulation, as well as a more careful toilet of the wound.

A 1 per cent. procain solution with a few drops of epinephrin chlorid was used and complete anesthesia was produced in less than ten minutes, which was a saving in time over general anesthesia. The infiltration of the scalp so reduced the bleeding that when the excision was made much time was saved in that only the larger vessels required clamping, and very often the field was bloodless. The hemostatic effect of the infiltration lasted always through the operation, or sufficiently long for clotting to take place in the constricted vessels, and in no case in this series did later hemorrhage occur. So satisfactory was local anesthesia in head operations that when there was a complication of other wounds the head operations were done under local anesthesia, and a general anesthesia given later for the debridement of other wounds. This applied not only to work at the front where complete operations

were done, but it proved to be just as successful at the base hospitals where operative procedures were done for brain abscess and other infective conditions associated with retained foreign bodies, cerebral hernia, fungus, etc. Another great advantage in the use of local anesthesia was the possibility of cooperation by the patient in asking him to blow his breath or cough; by thus increasing intracranial pressure it was possible to quickly remove the disorganized brain tissue, blood clots and often foreign bodies. In brain abscess it had proved most valuable, not only as to location but as to the area through which it might be approached.

Local anesthesia was *par excellence* the method of choice in cranial surgery. There was no pain in the bone, the dura was insensible to cutting though it would not stand traction or rough handling, and the manipulations of the brain itself never reached the threshold of consciousness. The operative technic, however, was of the greatest importance. After shaving the head and making as complete a neurological examination as possible, the following procedure was adhered to by Neurological Unit No. 1; complete excision of the scalp wound, avoiding contact with the lacerated edges; removal of the bone injury *en bloc*; evacuation of disorganized brain substance by having the patient blow against his closed lips or by coughing and also by catheter suction and irrigation; the detection of foreign bodies and bone fragments by catheter palpation and, after their removal, the instillation of dichloramin-T; and primary suture of dura and scalp. If the scalp defect was too large to permit suture without tension, the defect was covered by some plastic procedure. This was in many essentials the technic advocated and used by Colonel Cushing. In ventricular penetrations it was found possible, after removing the disorganized brain substance, to remove foreign bodies from the ventricles by direct inspection, using small retractors.

Observations had been made on a series of seventy-nine cases, thirteen of which were fractures with intact dura, the remaining cases representing all degrees of brain injury associated with lesions of venous sinuses, ventricular penetrations, and combined lesions with frontal sinus or mastoid complications. Nine of the thirteen fractures with intact dura were complicated with either extradural or intradural hemorrhage, producing compression symptoms of varying degree. The total mortality was represented by five deaths. Judging from reports on hand and from many cases personally examined by Major Ney, he did not believe that the late complications would be many. In the examination of about 200 cases not a single one of abscess or cerebral hernia had been observed in any having had the complete early operation.

#### DISCUSSION

DR. ALFRED S. TAYLOR expressed his appreciation of the privilege of listening to Dr. Ney's splendid paper in which there were two things that impressed him particularly: the very great interest of the intrinsic material of the article, and the complete lucidity of the presentation of a complicated and difficult subject. The application of cranial surgery as performed in the army to civil life was plain, and it was also clear that one should apply local anesthesia more than one had been accustomed to do.

DR. HAROLD NEUHOF said that Major Ney had presented the best results that had been obtained in gunshot wounds of the brain of the American Expeditionary Forces. The earlier mortality figures in dural penetration were about 50 per cent. With the improvement in technic, as advocated chiefly by Cushing, this was reduced to about 30 per cent. and most of the surgeons

were satisfied to have it at that low figure. Major Ney's was therefore a remarkable achievement. The technic he described was one that was followed, with a few variations, by all the teams under Cushing's command with parallel results. The principle had been to permit dural defects, even if large, to remain as such. Dr. Neuhof himself believed that some form of dural repair was indicated and, when the tear was too large, he employed transplantation of fascia. Fascia lata was employed and entered into the dural defects. The results were satisfactory, both immediately and as later reported. Dr. Neuhof stated his belief that local anesthesia for operations on the head not only proved the method of choice for war wounds but would similarly prove the anesthesia of choice in head operations in civil life. Since his return he had done several operations on the head under local anesthesia, among them a bilateral suboccipital craniotomy, as well as an osteoplastic flap in the parietal region, with results that encouraged its further use in head surgery.

COL. EDWIN BEER added a few words from his personal observations confirming Major Ney's statements. He declared that there was no doubt that these results were the best attained, but whether credit was due to Major Ney's own skill than to the use of local anesthesia there was some question. Dr. DuMartell of Paris told the speaker that he did all his civil skull and brain surgery under local anesthesia and had even removed a cerebellar pontine tumor, which was a difficult procedure. The most remarkable thing, proved by Ney and others, was the discovery that the brain was able to stand so much contamination without disastrous effect. One could not possibly get absolute sterility in these war wounds, and yet the dura could be closed as well as one could close up the knee joint, showing that the vital processes could be trusted to cope with the residue of infectious material which could not be removed.

DR. SACHS considered that the two most impressive points about this address were: First, the observation that local anesthesia acted as a hemostatic of the scalp; he had often thought that the large number of deaths in children following cranial surgery was due to loss of blood. Secondly, he was interested to learn that the surgeon's finger was not in future to be brought in contact with the brain substance and hereafter only the catheter would penetrate the depths of the brain. It would also be a great aid to the recognition of the presence of abscess on the operating table if the patient himself under local anesthesia could assist by exerting intracranial pressure through blowing out his cheeks, indicating the exact site and thus doing away with so much of the indiscriminate puncturing of the brain tissue that had been necessary in times gone by. It had been a delightful privilege to listen to this clear exposition of such valuable experiences and remarkable results as Major Ney had attained.

## Book Reviews

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THE AUTONOMIC FUNCTIONS AND THE PERSONALITY. By EDWARD J. KEMPF, M.D. Nervous and Mental Disease Monograph Series, No. 28, 1918.

A new viewpoint will be given to many readers by this monograph. In it psychiatry is found looking toward physiology for help, but modestly suggesting that it has knowledge of its own to give in return.

The first part of the book is given over to establishing a great reflex arc with the whole autonomic apparatus as its afferent arm and the cerebrospinal nervous system as a proficient arm. Experimental work (Cannon) has shown gastric contractions are concomitant with hunger pangs. Using this and other physiologic facts (Sherrington, Crile) as a basis, the author claims that emotion comes into existence only as peripheral autonomic reactions become aroused. Cannon's statement that hunger "may take imperious control of human actions" is given as the key to the dynamic functions of the personality, taking for granted that all autonomic cravings have the same physiologic function as hunger. The efferent side of the great arc of the cerebrospinal system is to be considered only as an agent developed and used by autonomic apparatus to get this or that subject in order to satisfy (neutralize) an effective craving. Hunger drives the brain and its striated muscles to get food. Thus "the mind" is brought down from its high throne and scattered among a lot of lowly organs.

The functioning of this great reflex arc as it deals with external stimuli in differing accidental associations (the conditioned reflex of Bechterew) determines the acquired traits of the personality and the symptoms of mental disease. Into his own psychobiologic terms the author goes on to translate the *libido* and other psycho-analytical conceptions, the higher emotions, memory, consciousness and life in all its aspects.

In a study of tonus in Part 2 the book best deserves the attention of the psychiatrist. After a consideration of "postural tonus" (Sherrington) and its dependence on the continuing activity of many reflexes, it is argued that it is foolish to consider that there is no emotional state when the individual shows no perturbation in his behavior. "An effective status continually exists." It is noticed that postural tonus varies with the affect as when one drops a lightly held razor on hearing surprising news. An affective craving which cannot find satisfaction through the cerebrospinal system is seen as stored in a heightened postural tension of some viscus. Attention is called to the apparently unchanged muscle tonus of catatonia and of agitated melancholias.

In a sense the important things described in this book are true whether the contentions in it are later upheld or not. The writer has described at least one side of a fundamental situation, an achievement worth while.

NEUROLOGICAL CLINICS. Edited by JOSEPH COLLINS, M.D. Paul Hoeber, New York.

This book is made up of 271 pages. It consists of a series of case reports with the discussion chiefly concerned in the diagnosis and differential diagnosis. The cases recorded are for the most part of cerebral or spinal origin and are

derived from the first division of the Neurological Institute. Collins is aided in his work by the various members of his staff.

The cases reported are practically all of organic nature. They are well presented, and the discussion which is incident to each case is very often illuminating.

The reviewer was made "tres triste" by a remark on the first page made by the editor. Collins states very naively that the diagnosis of the brain is largely a matter of guesswork. Then he proceeds to diagnose his case very skilfully and with an acumen that belies the foregoing remark.

CESARE LOMBROSO. *Storia della vita e delle opere narrata dalla figlia.* Turin, 1915.

In the volume before us Signora Ferrero has told, with much sympathy and natural appreciation, but with good taste and restraint, the story of her father's long struggle for recognition against prejudice and a curious opposition.

Cesare Lombroso was born of a well-to-do and eminent Spanish-Hebrew family in Verona in 1835, but soon after his birth the family met with financial reverses and his means became limited. His earlier education was subject to Austrian tyranny. He was obliged to go to a school under Jesuit control, and under such surveillance that a comrade once reported to the police his possession of a copy of Lucretius. An ardent and precocious student, fond of literature, Lombroso, after the reopening of the universities, entered the school of medicine at Pavia, at the age of 17, and from thence, three years later, he went to Vienna. His mind was filled with a desire to benefit his fellows by study of the treatment of some of their more distressing ills, and to do for his countrymen what was being done for the people of Vienna, especially in the treatment of mental diseases. To that end he began a study of cretinism in Lombardy, making important contributions to the etiology and treatment of that affection. On his return to Verona the political situation led him to enter the medical service of the Piedmontese army, and after the close of the war he was transferred to the division hospital at Pavia, where he again began to turn his attention to mental diseases and to study especially the relation between the insane and the criminal. In 1864 he left the army to devote himself more especially to the study of mental disease, but returned for the war of 1866. After finally leaving the army, he took up the study of pellagra, at that time extremely prevalent in Italy. Lombroso claimed that pellagra was due to a toxin formed in the perisperm of spoiled maize, and that it could be successfully combated by arsenic and by a regulation of the diet, excluding maize. His studies were presented in 1870 to the Lombard Institute, which offered a prize for a memoir on pellagra of definite benefit to society. The Institute, while highly commending Lombroso's work, failed to award him the prize, which his work apparently merited. In 1871 he was appointed to take charge of a hospital for the insane at Pesaro; but, owing to the lack of students and the absence of facilities for teaching, he soon returned to Pavia. In the meantime, Lussana, professor of physiology at Pavia, had attacked Lombroso's doctrines in regard to pellagra. In 1874 he accepted the chair of legal medicine, not including mental diseases, at Turin. Soon after, the Lombard Institute again took up his claims as to the origin of pellagra, and a most acrimonious attack followed. It is difficult to understand the animus which led to the attack on Lombroso's doctrines, and the apparent unfairness which actuated his opponents. But the story, as told by Signora Ferrero, shows a disregard of evidence strange in scientific men,

although she does not fully make clear the reason for the virulence with which he was attacked. Lombroso's experiments were confirmed by other observers outside of Italy and in time gained acceptance even in Italy itself, and it is only of late years that further research has thrown doubt on some of his theories. In 1875 he went to Turin as professor extraordinarius in legal medicine instead of in psychiatry, with scanty facilities for his anthropologic and criminal museum, without the clinical facilities of a hospital for the insane, and with merely such clinical facilities as were furnished by the prison of Turin, of which he was made physician. The following year he published his *Trattato antropologico sperimentale dell' Uomo delinquente*, which while meeting with marked success elsewhere in Italy, in Turin was received with much the same disfavor as his work on pellagra. The following year he published a new edition of his *Genio e Follia*.

In the meantime he had married and had several children. For several years his life at Turin was a hard one. With scanty means, inadequate facilities, unsatisfactory clinical advantages, and lack of recognition, he struggled on, in hope of ultimate triumph, declining a better position at Reggio-Emilia because there he would have no teaching opportunities. A second edition of his great work, now called simply *L'Uomo Delinquente*, was called for and attracted attention all over the world. In the early eighties he started the *Archivio di Psichiatria*. His views on pellagra began to meet with more general acceptance and when the first International Congress of Criminal Anthropology was held Lombroso was accorded wider recognition outside of Italy. In spite of this foreign recognition, the new penal code of Italy, put forward in 1888, failed to adopt the chief tenets of his teaching, and the second Congress of Criminal Anthropology, held in Paris in 1889, showed considerable opposition, Manouvrier claiming that although there was truth in Lombroso's system, the system as a whole was false. In 1891, however, Lombroso was appointed to the full professorship of clinical psychiatry in Turin, to succeed Morselli. He was also made inspector of asylums. A short time later, in conjunction with Tamburini, he examined Eusapia Palladino and failed to expose her trickeries. In consequence of his investigations, he openly espoused spiritualism, writing a treatise in its defense. In 1899 a law was passed dealing with pellagra and adopting, in the main, Lombroso's theories. From that time on he led an active life at Turin, writing a great deal, upholding his own views, and receiving many honors as the pioneer of criminal anthropology, although the controversies over his views continued until his death in 1909.

Writing as a devoted disciple of her father's doctrines and as a zealous advocate of his views, Signora Ferrero fails fully to make clear the motives for the opposition to Lombroso or set forth the arguments against his teachings. Consequently, it is difficult for a foreigner to understand the motives back of the opposition and to comprehend the apparent unfairness of many of the polemics against him, so that in reading this story of Lombroso's struggles and the contests that embittered his life almost to its close, the natural query is whether her filial partisanship has not led her to give a somewhat prejudiced picture. Like all pioneers, Lombroso was an enthusiast and had the imaginative temperament of the poet rather than the cold judgment of the scientist, although the latter was not lacking. Hence we find in *L'Uomo Delinquente* not only the exact and arid measurements of the criminal but also many speculative theories as to the identity of the *criminale nato*. In his earlier years Lombroso was a disciple of homeopathy, in later life

he endorsed Eusapia Palladino and spiritualism. These tendencies, therefore, may have aroused a spirit of distrust in and criticism of the scientific value of his work. It must also be remembered that the teachings of *L'Uomo Delinquente*, in accord with the determinism of modern science, were well calculated to arouse not only the odium theologicum, still most potent in his earlier years, but also the odium juridicum fully as potent and still pregnant. Although many of the subordinate teachings of *L'Uomo Delinquente* have not been confirmed by later study, nevertheless, Lombroso was a great pioneer; his main teachings in criminal anthropology have won general acceptance, and in his day and generation in that field he must be hailed *il maestro di color chi sanno* (master of color).

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